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CARDIOVASCULAR DAMAGE RESULTING FROM NONPENTRATING STEERING WHEEL IMPACT

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DEDICATION

This dissertation is dedicated to the finest man I have ever known; a man, who for more than a quarter of a century has served his patients and his community untiringly, never once faltering, even in personal illness, yet asking nothing for himself.

A man of humble origins, left motherless at an early age, who, in the face of seemingly insurmountable odds, sought for and attained his goals.

A man of unquestionable character ... a man of kindness and compassion ... a man of limitless charity ... a man of unusual goodness ... and in my eyes, a man of indisputable greatness ... my beloved father.

The Author

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

In many automobile accidents the driver receives blunt trauma to the thorax when he is thrown forward at impact and strikes the steering wheel.

Physical injuries often appear minor and the associated discomfort, being generally expected, may cause other symptoms to be lightly dismissed or perhaps totally ignored.

Immediate attention in the emergency room is focused on the more dramatic injuries - wounds are cleansed, lacerations sutured, broken bones set. Little attention if any is given to possible cardiac involvement, especially in the absence of specific symptoms. X-ray studies are routinely ordered on accident victims with chest pain, and thoracic trauma is often ruled out in the absence of broken ribs.

Damage to the heart is rarely, if ever considered, and this is unfortunate because cardiac injuries probably occur far more frequently than now suspected. With severe impact, subsequent congestive heart failure, myocardial contusion and myocardial rupture have been observed. Less severe impact has resulted in transient and reversible indications

of cardiac damage.

The complexity of the diagnostic problem is further complicated by the asymptomatic patient, and the unfamiliarity of the attending physician with such conditions. The fact that the patient usually recovers may account for the lack of more meaningful knowledge about cardiac injury.

Prior to the inception of this study, numerous case histories of patients who sustained nonpenetrating cardiac injuries were reviewed. The salient features of many of these histories are that (a) damage to the heart may not be recognized immediately because of other, more dramatic physical injuries, (b) the temporary nature of some particular "trauma related" cardiac disorders, and (c) the reversibility of other, more severe, cardiac irregularities.

It was, perhaps, tenuous to embark upon a project which sought to elucidate a "potential cause-effect" relationship between two entities such as steering wheel impact and cardiac damage. However, it is the considered opinion of this author that, while the evidence and conclusions reached herein, may not be fully accepted by all readers, the proposition is, nonetheless, reasonable and the methodology both logical and practical.

Heart disease may manifest itself in many unusual forms and for many unusual reasons, not the least of which is trauma, and Burchell (1) has astutely characterized the state of the art when he said "... and always with a heart contusion, arise both doubt and much confusion...."

Types of Injuries

Cardiac injuries are most frequently caused by crushing of the

thorax or blows to the anterior chest wall.

Barber (2), in reviewing autopsy material from persons subjected to blunt thoracic trauma, postulated that children and young adults who had no previous history of heart disease might be predisposed to cardiac damage. He concluded that fractures of the ribs and/or sternum need not occur before the heart may be damaged, because in younger persons with highly resilient thoracic cages the blunt forces would not be distributed evenly throughout the bony framework of the chest but, rather, con entrated into the soft thoracic tissue.

Moritz (3) pointed out that, during the first five decades of life, the thoracic cage is, as a rule, sufficiently plastic enough to permit great distortion without fracture, and this fact is of primary importance to a consideration of cardiac injury resulting from blunt trauma. Impact may not damage the ribs or sternum, but severe injury may result to the intrathoracic viscera. Arenberg (4) studied a series of nonpenetrating injuries to the chest. One hundred sixty eight resulted from falls, 38 resulted from impacting a swinging object and 28 resulted from a squeezing or crushing force. He observed that the incidence and severity of cardiac damage was less in persons who had suffered broken ribs than in those whose thoracic cage was unbroken. Thoracic rigidity, therefore, seems to be an important factor in protecting the heart and great vessels against damage from blunt injury.

Many nonpenetrating cardiac injuries are not recognized immediately unless the heart is ruptured and death occurs within a relatively short time. Hamilton (5) reported heart rupture in a child resulting from a fall. Bilderbeck (6) reported heart rupture in a young adult who

fell, but post mortem examination revealed no other evidence of trauma. Bright and Beck (7) studied some 200 cases of cardiac damage resulting from nonpenetrating chest injury. About 90 per cent of these displayed fatal heart rupture. It might be concluded that the heart is incapable of withstanding severe trauma; however, the authors were quick to point out that the majority of nonpenetrating wounds are not recognized clinically, since the degree of damage may be subtle or delayed in appearance. Barber (8) reported a case of cardiac damage resulting from trauma which was alleged to have occurred 17 years prior to diagnosis. This report lends credence to the contention that trauma related heart damage may not be discovered for long periods of time. Historical evidence, in general, comes chiefly from traffic and industrial accidents. Howat (9) points out that the mechanisms of injury that occur when a heart is ruptured as the result of a fall are not fully understood. Beck (10) analyzed a large number of traumatic heart ruptures and concluded that in accidents in which the legs and abdomen are suddenly buried, the heart may rupture from indirect violence due to internal pressure.

Saphir (11) recorded a case of heart rupture from indirect trauma in a child 4 years of age who was run over by a car, and Stephens (12) reported the case of a youth whose heart was ruptured as the result of the abdomen being crushed. An analysis of 152 cases of fatal heart rupture by Bright and Beck (7) indicated that no single chamber of the heart appears to be more susceptible to rupture, as the result of blunt trauma, than any other chamber. In this series of cases 11 patients sustained rupture of the interventricular septum and 76 fatalities were attributed to heart tamponade resulting from hemopericardium. Leinoff (13) in

reporting the results of post mortem examinations on 50 nonselected automobile fatalities stated that in one instance, all four chambers of the heart were ruptured.

Joeffe (14) investigated the pathological results of rapid deceleration into lap type seat belts. He suggested that cardiac damage was produced by the blood flow being suddenly blocked in the abdomen in both the vena cava and the abdominal aorta. The effect produced is similar to that of a closed liquid system subjected to forces from above and below. The heart is squeezed between the two forces and stretched in a transverse direction.

Seat belts may cause another type of cardiac injury. When rapidly decelerated, the body, acting as a second class lever over the belt, flexes forward causing the chest to strike the steering wheel with extremely high force. In Joeffe's experiment, lung congestion was noted during rapid deceleration into seat belts; however, this either diminished or completely disappeared within a fraction of a second after the force was applied. Even so, it is possible that the hydrostatic forces transmitted to the heart by way of the great vessels may increase the bursting tension within that organ to the point of rupture (15), or produce cardiac pathology by momentarily stretching the muscle beyond its limits of distensibility (14). Additionally, sufficient force may cause permanent deformation of the myocardium (14), rupture of the intrapericardial portion of the thoracic aorta, laceration of the valves or explosive disruption of the walls of the left ventricle or left atrium, or otherwise cause some type of cardiovascular damage (3).

Roberts, Moffat and Berkas (16) disagree with the concept that

hydrostatic forces will cause cardiovascular damage. After experimentally subjecting mongrel dogs to blunt thoracic trauma, they concluded that pressure surges through the vascular system are not responsible for the tearing of vessel or chamber walls. It should be pointed out that anatomical differences between dog and man such as the shape and structure of the thorax, the shape and size of the mediastinum, and the number of great vessels branching from the thoracic aorta renders the validity of any extrapolations from dogs to man questionable or tenuous at best.

Cardiovascular Disorders Occasionally Attributable to Trauma

Barber (2) has stated that both angina of effort and coronary thrombosis possibly arise as the direct result of trauma, however the clinical features of these conditions may be indistinguishable from the disease which results from natural causes.

Blackall (17) described five cases of angina pectoris and wrote of the last one:

> The following as an instance not common, of the disease being connected with external violence, will perhaps be deemed worthy of the attention of the reader. R. B. aetat. [sic] 60, a coachman. Three months before he consulted me received a violent blow, from the pole of a carriage, which forcing him against a wall caused some little contusion of the surface of the chest, and an internal pain for which he was blooded, and used a liniment. He continued affected in his breathing on motion and two months afterwards, whilst he was pitching some hay into a loft he felt a violent pain in the region of the heart spreading rapidly to the left arm, with faintness and inability of movement. A similar paroxysm came on several successive nights waking him with an approach to deliquium and great anxiety. It continued to return on every exertion, and went off only by rest. But after he had quitted his situation, and was liable to less fatigue, the attacks became milder.

Modern technology, traffic and mechanization of industry have increased the liability to accidents for all sections of the community,

and this may be part of the reason why the relation of angina to injury has received relatively little consideration. Barber (8) reported the case of a man who fell on his chest. Some two weeks following the incident there was anginal pain which recurred over a 3-month period. It gradually subsided and the man resumed his normal activities. Campbell (18) described the case of a 47-year old man who was struck near the left nipple by a piece of heavy furniture which pinned him against a wall. Two hours after the accident he experienced anginal pain which continued intermittently for a period of 7 years. Warburg (19) in a study of 59 traumatic heart lesions reported 16 cases of clinical angina.

Case reports of coronary thrombosis following trauma to the chest are not unusual. Wearn (20) described the case of a 55-year old man who was struck about the chest by a large heavy object. He collapsed following the injury but soon regained consciousness. Several days later he experienced a typical attack of coronary thrombosis. Starling (21) reported a case of coronary thrombosis in a 60-year old man occurring 16 days after a fall down some stairs, in which the chest was bruised.

Leinoff (13) reported a case of coronary thrombosis in a swimmer developing 24 hours after being struck in the chest by a large wave. In every case reviewed and reported the typical episode usually occurred a few days following trauma. As will be pointed out in the discussion of myocardial contusion, bruising near a diseased coronary artery might lead to occlusion, but there remains the likelihood of coincidence. Whether or not coronary thrombosis may result from blunt trauma to the chest is still questionable, but the possibility cannot be lightly dismissed.

Consideration must be given to the clinical course of persons

who suffer coronary heart disease, and sustain some type of chest injury. Arenberg (4) in studying some 250 men who came under observation following injury, noted that it was in those patients with hypertension or coronary disease that he found heart disability. Whether a relation existed between trauma and advanced heart disease cannot be proven, but there appears to be justification for his contention that some might have been saved from symptoms if the heart condition had been considered earlier.

Disorders of Rhythm

Warburg (22) in reviewing 225 cases of nonpenetrating injuries to the heart described a variety of arrhythmias including auricular flutter and fibrillation. Price (23) and White (24) have stated that auricular fibrillation may develop as the result of trauma. Khan and Khan (25) described dysrhythmia following trauma, however the patient was known to suffer from mitral valve disease. They concluded that the signs and symptoms of cardiac dysrhythmia should develop immediately after trauma in order to be consistent with the cause and effect relationship.

If auricular fibrillation follows direct violence to the thorax, the possibility arises that the heart has been contused. Kissane (26) reported two such cases, in which a tendency for congestive failure persisted in both instances. Warburg (22) and O'Farrell (27) described similar cases resulting in death. The fact that contusion may have occurred in addition to the auricular fibrillation requires careful observation and a period of rest before assessing the extent of damage to the heart or considering the problem of restoring normal rhythm when the arrhythmia results from blunt violence (2).

Levison (28) reported auricular fibrillation, in the absence of any other disability, in a 20-year old man following crushing injury of the chest. Auricular fibrillation may also result from non-thoracic trauma. For example, cases have been reported following electric shock (29), and the inhalation of noxious gases (30, 31).

Auricular Flutter

Although of similar etiology, auricular flutter is less common than auricular fibrillation. Khan (32) described the case of a 59-year old man who received a direct blow to the chest. Approximately 3 weeks following the injury an auricular flutter was recognized which persisted until death occurred 4 months later. DiPalma and Schultz (33) stated that auricular flutter originates not because some area of the heart is hyperirritable, but because of the presence of one or more depressed areas having relatively prolonged refractory periods and even more prolonged conduction rates. These areas of partial block, which may be very small, allow reentry of the impulse previously generated by some other region or previously passed by the blocked area, and thus appear to become rapidly discharging foci. Blunt trauma to the heart may easily depress a conduction pathway, resulting in auricular flutter.

Paroxysmal Tachycardia

Barber (2) observed two cases of paroxysmal tachycarida resulting from blunt thoracic trauma. The attacks in one case ceased after approximately 4 weeks; a 41-year old male hospitalized for fractured ribs and hemothorax developed nodal tachycardia 72 hours after injury. Electrocardiographic studies showed that the P wave and the R wave occasionally coincided, but more frequently, the P waves were inverted and

followed the R waves. The condition persisted for approximately 2 weeks. Kissane, Fidler, and Koons (34) reported the case of a 42-year old man who fell and fractured two ribs. Three days later he experienced severe dyspnea and an electrocardiogram revealed nodal tachycardia. Autopsy revealed hemorrhage into the mediastinum and myocardium.

Sinus Bradycardia

A distrubance to the sinus node will result in a slow sinus rhythm and a heart rate below 60. Barber (2) has reported four cases of sinus bradycardia which resulted from direct violence to the thorax. Three of the cases in this series were detected by routine electrocardiograms. Two of these patients were asymptomatic; however, the third experienced syncope approximately 1 hour following steering wheel impact. The fourth case was seen some 5 days after receiving a severe blow to the chest in an athletic contest. The only other symptom in this series of patients was a relatively low blood pressure. Sinus bradycardia has been reported in boxers by Jokl (35). Barber (2) quoted Kulbs who reported the case of a 16-year old boy, who a few hours after a fight, complained of chest pain. Bradycardia was the only symptom in this youth.

Heart Block

Heart block of traumatic origin does not appear to be common. Barber (2) reported that the injuries involved are somewhat similar to those leading to delayed rupture of the heart, and essentially it is a form of myocardial contusion. Heart block leading to death has been noted in some seriously damaged hearts; however, there are a number of records of heart block as the only obvious disability remaining after

injury. Arenberg (4) recorded heart block persisting for 20 months after direct violence to the chest of a 42-year old man. Tuohy and Boman (36) recorded traumatic heart block which changed to a bundle branch block and finally to a complete block. Barber (37) reported the case of a youth who received a crushing chest injury, and heart block was revealed upon routine electrocardiogram. Follow-up reports indicate that the functional capacity in cases in which the disability persists is reasonably good (2).

Extrasystolic Arrhythmia

Barber (2) believed that the significance of premature contraction is not well enough defined to make it a profitable study in relation to trauma. Arenberg (4) reported the case of a 31-year old man who fell 10 feet and struck his chest on a stone floor. Following the accident he complained of "palpitation and fluttering" but remained at work. Six weeks after this incident he was examined and extrasystoles were the only notable abnormality. This condition subsided within 3 months. Leinoff (13) reported the case of a 21-year old man with numerous nodal extrasystoles following a crushing injury. Despite some referred pain and occasional dyspnea, the patient recovered.

Ventricular Fibrillation

Ventricular fibrillation probably represents the occurrence of a circus movement of excitation in the ventricles, and in man is usually fatal unless the process is stopped by counter-shock. However, instances of transient recurrent attacks of ventricular fibrillation have been reported in patients without organic heart disease (38, 39). Barber (2)

pointed out that if immediate death results from a direct blow over the heart, and there is no post mortem evidence to account for the fatality, ventricular fibrillation is a reasonable explanation.

Types of Heart Damage

Moritz (3) has categorized cardiac injury caused by the application of blunt violence to the thorax into three groups according to the severity of the disruptive changes: (a) commotion, (b) contusion, and (c) laceration. When there is a disturbance in cardiac function that has been caused by impact or agitation of the heart without the production of gross or microscopic evidence of injury, cardiac commotion has occurred. Moritz (3) quoted Kulbs who pointed out that functional disturbance caused by an impact to the heart may be disproportionate to the morphological evidence of injury. Moritz (3) stated that a precordial impact may result in a severe and even fatal disturbance in the function of what may appear to be an undamaged heart.

Beck (10) in a study on heart trauma reported that the object responsible for fatal precordial impacts may not be immediately recognized. Moritz (3) pointed out:

> Without the benefit of a direct examination of the injured heart, the clinical observer has no way of recognizing the extent to which the observed functional disturbance may be accompanied and explained by disruption of structure. Usually the most that can be said on the basis of clinical examination and medical history is that cardiac dysfunction developed immediately after, and presumably as the result of external impact. A post-traumatic functional disturbance of the heart that is transient does not indicate the absence of structural change; neither does one that is fatal require the presence of a visible structural lesion.

It is rarely possible to make a direct examination of the heart of a living person who has recently suffered a nonfatal cardiac injury,

and therefore the frequency with which such transient post-traumatic disturbances occur independently of structural lesions is not known (3). Bright and Beck (7), and Barber (2) have documented cases of transient post-traumatic disturbances in cardiac function in man following impact to the chest, and there are a few reported instances of death resulting from heart failure following blunt injury of the chest, in which there was a conspicuous absence of gross or microscopic evidence of injury (2, 22).

Gore (40) reported the case of a young soldier not previously recognized to be suffering from heart disease. He received a severe blow to the precordium, which was followed by pain, shock, and tachycardia. Four days after the injury, the heart went into a "gallop" rhythm, and precordial friction rub was heard. Death from an apparent ventricular fibrillation occurred 7 days after the original injury. An autopsy revealed that death was caused by subacute and chronic myocarditis which had been present at the time of injury but had remained asymptomatic until the traumatic episode triggered the mechanism which resulted in death. This is an excellent example of the unreliability of the clinical diagnosis of traumatic heart disease.

Moritz (3) has defined cardiac contusion as a diffuse extravasation of blood into the interstital spaces of the heart muscle, caused by impact. Minute vascular defects through which blood escapes may be created by excessive distortion or stretching of the tissue or by a sudden rise in intracapillary pressure because of the hydrostatic effects of sudden compression (3).

Khan (41) reported the case of a 17-year old youth who received

a myocardial contusion following a nonpenetrating chest injury in an automobile accident. The case is cited because the immediate physical findings were suggestive of pericardial injury and the electrocardiographic abnormalities, at first, resembled acute epicardial or pericardial injury, which later were more characteristic of myocardial damage, specifically contusion. Moritz (3) pointed out that if only the heart is examined it may be difficult to distinguish between contusion and certain nontraumatic interstital extravasations of blood, however, he further stated that if an interstital extravasation of blood represents a true contusion, it should be localized to the heart and such other structures as were in the path of force, because bleeding which was spontaneous and resulted from a systemic disorder will not usually be confined to the thoracic viscera.

Laceration is a gross defect in the continuity of tissue caused by a crushing or stretching force and, although it may not be associated with contusion, it is invariably associated with hemorrhage (3). The crux of this lies in the fact that at first glance it may be impossible to distinguish between traumatic rupture of the heart and a spontaneous rupture from disease. Moritz (3) has stated that, while the heart may be ruptured by a precordial impact, spontaneous rupture at the site of a recent myocardial infarction occurs more commonly. Helpern (42) reported that he had never seen contusion, or laceration, of the human heart or coronary arteries as an isolated injury, and he believes that, in the absence of evidence of external cardiac trauma, cardiac lesions are probably nontraumatic.

Beck (10) has emphasized the importance of the steering wheel

impact in which the driver is thrown forward by the sudden deceleration of the vehicle, however as previously discussed, it should be borne in mind that the object responsible for the fatal precordial impact may not be immediately recognized.

Cardiac contusions and lacerations resulting from blunt injuries may be anterior and directly beneath the external site of impact or may be remote from it, however injuries from anterior thoracic trauma are sometimes found on the posterior aspect of the heart muscle, presumably from compression of the heart against the vertebral column. Moritz (3) quoted Urbach who reported the distribution of cardiac lesions resulting from blunt trauma in the following order of diminishing frequency: right atrium, left ventricle, right ventricle, left atrium, interventricular septum and valves.

It has been observed that blunt injury to the heart may lead to a wide spectrum of myocardial damage, especially hemorrhage in the absence of laceration, and Moritz (3) concluded that these are apparently the result of minute focal lacerations of muscle and probably result from the impact being delivered while the ventricles are filled with blood. Warburg (22) reported several cases of this type, and has stressed the frequency of this type of injury in human beings.

Pericardium

Barber (2) pointed out that a pericardial friction sound as evidence of heart trauma had long been recognized in clinical experience. The actual time of onset following injury is widely variable. Smith and McKaown (43) reported the case of a 17-year old youth who received a blow to the chest in a motor accident. Pericardial rub was not detected until

the eighth day after injury. On the eleventh day an electrocardiogram suggested pericarditis. They concluded that the parietal and visceral layers of the pericardium had probably been bruised without any physical sign of friction, until the bruising became more organized. Barber (2) stated that evidence of pericarditis is an indication for complete cardiological evaluation, and that in the absence of underlying heart lesions, complete recovery may be expected. Fasola, Baker and Hitchcock (44) reported that following blunt trauma to the chest, the majority of damage seems to be confined to the myocardium and the pericardium. They interpreted experimental findings as suggestive of coronary artery occlusion and pericarditis. Moritz (3) and Helpern (42) disagree with the belief that pericarditis may result from blunt violence to the heart. Helpern stated that there was no convincing pathological evidence of such an entity. He believed that the escape of blood into the pericardial sac may cause pericarditis but, if blood escapes into the pericardium as a result of blunt violence, it does so because a chamber has been lacerated, and the patient rarely if ever survives such an injury for a period long enough to permit the development of pericarditis.

Hemopericardium

Barber (2) believed that hemopericardium is the most important pericardial condition for consideration in connection with nonpenetrating wounds of the heart. He stated that small amounts of blood may account for some of the symptoms of myocardial contusion; however, with hemopericardium, signs and symptoms of cardiac tamponade develop. Shock, hypotension, low pulse pressure and bradycardia are often noted.

Tamponade

As observed by Moritz (3) penetrating injuries of the cardiac chambers or of the epicardial vessels are usually characterized by relatively slow bleeding, whereas hemorrhage following the type of cardiac laceration characteristically caused by blunt impact is usually so rapid as to be fatal almost immediately. Moritz further states that if the immediate functional effects of the injury are survived, and within a week the interstital extravasation of blood is absorbed with little or no residual abnormality, myocardial contusions generally heal. The persistence of functional disturbances for more than a few days after injury, should, in the absence of hemopericardium, cast doubt on the traumatic origin of the disturbance. Cary, Hurst and Arentzen (45) pointed out an important exception to this generalization which is the traumatically produced laceration of the interventricular septum. They stated that such an injury may result in chronic, progressive cardiac disability similar to that caused by a congenital interventricular septal defect.

Myocardium

Barber (2) concluded that myocardial rupture may occur immediately after blunt injury to the heart or on rare occasions be somewhat delayed. Groom (46) reported the case of a 16-year old boy who was found dead, 1 month after being crushed against a railing. Although he had been bedfast for 5 days following the incident, there was no external bruising on the chest wall. An autopsy revealed a ruptured left ventricle. Barber (2) in reviewing case histories of 12 delayed heart ruptures found that 66 per cent of the victims were children or young adults having elastic thoracic cages. Most of these had sustained a crushing type

injury which had been dismissed or nonsevere. Priest (47) reported the death of a youth 2 days after being struck in the chest by a hard ball. This sudden death was due to rupture of the right ventricle. The youth was asymptomatic prior to death, which is illustrative of the symptomless nature of the lesion which was also common in each of the 12 cases reviewed by Barber. None of the victims in Barber's series had suffered any apparent heart damage. Tuohy and Boman (36) reported the case of a 63-year old man who received a blow to the chest from the steering wheel of an automobile. Two weeks after the accident he died and an autopsy revealed a rupture of the left ventricle. Glendy and White (48) recorded the case of a 24-year old man who was also injured in an automobile accident. The heart appeared normal; however, surgery was performed for removal of a ruptured spleen. The man died shortly after the procedure and an autopsy revealed a ruptured anterior papillary muscle in the left ventricle, and a tear which extended 1 centimeter into the myocardium. Barber (2) stated that because delayed rupture of the heart is not common, one might be tempted to believe that the essential lesion is most likely to tear in the endocardium. Such a tear would be less likely to give rise to symptoms than would a contusion or partial rupture which involved the outer surface of the heart and myocardium.

Krumbhaar and Crowell (49) reported the fatal rupture of a traumatic infarction beginning in the outer part of the left ventricle in a 38-year old male. Prior to death there was some precordial pain, however no diagnosis was made until autopsy. In the series of 12 fatal cases reviewed by Barber (2) three patients died during the first week, three died in the second week and six survived for a period which varied from

3 weeks to 3 months. Bright and Beck (7) have suggested that the second week following injury is the most critical period during which the rupture is likely to occur. Barber (2) believed that after a crushing injury to the chest, it is a good practice to observe the heart for several weeks in attempting to determine the extent of delayed damage.

Contusion of the Myocardium

Khan (41) has been cited previously as reporting the case of a myocardial contusion in a 17-year old youth after steering wheel impact. In this case the patient complained of diffuse anterior chest pain, particularly left subclavicular and precordial. Chest films revealed no fractures; however, there was audible crepitus over the fourth and fifth left ribs anteriorly. The cardiac tones were of good quality without audible thrills or murmurs, and there was no enlargement of the cardiac silhouette. An electrocardiogram taken 24 hours after the accident suggested the likelihood of traumatic pericardial injury or myocardial contusion. Subsequent tracings with selected precordial leads revealed serial evolutionary changes resembling myocardial infarction. At no time did the tracings evidence arrhythmia or disturbance of intraventricular conduction. The chest pain gradually subsided and his recovery was uneventful. White (24) stated that contusion of the myocardium is probably fairly common and recovery seems to be the rule. Evidence from autopsy findings revealed to Barber (2) that small lesions compatible with recovery are common, and it seems probable that symptomless cases with recovery are by no means rare. He further stated that there is a high probability in patients with thoracic fracture, contusions of the lung, hemothorax or pneumothorax, that symptoms present might mask those

which arise from contusion of the heart muscle. In addition, bedrest which is necessitated by surgical lesions to other parts of the body may tend to keep in check symptoms referable to the heart. Lee, Ussher and Houck (50) reported the case of a 22-year old man who received a blow to the lower sternum while playing football. He was stunned but returned to the game within minutes. Almost 12 hours later he developed precordial pain, dyspnea, orthopnea, and constricting chest pain which radiated into the jaw. Physical findings were essentially negative, although a slight abnormality of the electrocardiogram was suspected, and some pericardial bleeding was not ruled out. Nine days after the incident he experienced tachycardia and severe dyspnea. Deep inversions in the T waves from leads I and IV of the electrocardiogram were observed 15 days after the accident. The dyspnea and chest pain were controlled by bedrest and the patient made an uneventful recovery. Barber (2) pointed out that particular emphasis need be given to the interval between the immediate reaction to the accident and the appearance of symptoms referable to the heart. Beck (10) described a case in which symptoms did not occur until 3 days following the injury, with death coming on the sixth day, due to myocardial contusion. Warburg (22) in discussing 51 cases of confirmed nonpenetrating chest injury, related that 15 had no symptoms which were referable to heart involvement. Apparently many of these accidents had serious associated injuries, making it difficult to analyze other symptoms. Barber (2) stated that in uncomplicated contusions a latent period seems to be the rule rather than the exception; however, when symptoms suggesting contusions are present, or certainly if the ECG is abnormal, not less than a month in bed is required.

Complications of Myocardial Contusion

Hemopericardium as a complication of myocardial contusion has been cited earlier (2, 7). Froment and Blanchard (51) reported the case of a 26-year old soldier who was struck in the chest by the recoil of a rifle. Hemoptysis developed some 10 days following the incident, and x-ray examination revealed an enlarged cardiac shadow. The man died 3 months later and an autopsy revealed definite signs of myocardial contusion.

Anderson (52) described a case of definite myocardial contusion, however an ECG simulated the picture of coronary thrombosis, and a systolic bruit and thrill suggested a lesion of the mitral valve or of the interventricular septum.

Sequels of Myocardial Contusion

Barber (2) believed that aneurysm is a rare but recognized sequel to myocardial contusion. Warburg (22) quoted Hilderbrandt in reporting the case of a 9-year old boy who fell down a stair case but did not fully recover. Autopsy at age 27 revealed an aneurysm of the left ventricle. O'Farrell (27) reported aneurysmal dilation of the lower aspect of the left ventricle in a man who developed auricular fibrillation following an automobile accident, and died three months later of congestive heart failure. Perry (53) in reviewing case reports of aneurysm related that in some instances actual trauma to the chest may be a precipitating factor for aortic aneurysm. He quoted Heller and Oppenheim in describing two cases of spontaneous aortic rupture, following a crushing injury to the chest.

Gore (54) stated that traumatic aneurysms of the aorta may result

from sudden forceful decompression of the thorax or from rapid linear deceleration such as may be experienced in falls from a height, or in motor vehicle crashes. Strassman (55) in reviewing 7000 medico-legal autopsies found 72 traumatic ruptures of the aorta; 11 had multiple tears, and of the 61 with only one tear, all but 3 were in the thoracic aorta and 38 were located just distal to the insertion of the obliterated ductus arteriosus. The second most vulnerable site was the first portion of the ascending aorta. Rice and Wittstruck (56) demonstrated that in linear deceleration of the thorax, the central portion, being the least rigidly bound, is snapped forward by the momentum of the body and the mass of the aortas blood content. Because there is more rapid deceleration of the proximal aorta which is fixed by the great vessels of the arch and the ligamentum arteriosum, great strain is exerted at its junction with the mobile descending segment. Similarly, the difference in fixation and mobility of the heart and the first portion of the ascending aorta may account for the vulnerability of the latter site. Gore (54) pointed out that in the absence of structural weakness, the rupturing force must be great; to avoid its dissipation along the vascular channel, it must also be sudden. Strassman (55) and Stryker (57) have reported incomplete forms of aortic ruptures demonstrated at autopsy, and stated that in persons who have died as the result of severe trauma it is not unusual to observe tears which are confined to the inner aspect of the aorta. Gore (54) believed that such lesions, occurring in nonfatal trauma may heal by fibrosis and scarring and represent a point of weakening; if sufficiently large and deep, stretching and herniation of the fibrous scar could result in formation of an aneurysm. Leonard (58) reported the case of a

dissecting aneurysm following a crushing chest injury, although this is, as pointed out, somewhat rare. Hollingworth, Johnston, and McCooey (59) reported four cases of saccular aneurysm of the aortic arch following severe chest trauma, all in young men who were followed for periods of a few months to 7 years. Steinberg (60) reported on five cases for periods ranging from 2 to 27 years. He noted that the sole fatality followed operative intervention for an aneurysm which had been asymptomatic for 5 years.

Gore (54) has suggested that clinical recognition of the condition requires a history of severe trauma involving the thorax, and radiological evidence of widening of the superior mediastinum in the vicinity of the aortic arch. Jackson and Slavin (61) pointed out the difficulty which may be encountered in the detection of aneurysms, especially those of the dissecting variety, in stating that only an angiogram can establish a definite diagnosis. This however is not always without a degree of danger and signs are not always present.

Coronary Artery Occlusion

Fasola, Baker and Hitchcock (44) stated that following trauma some electrocardiographic changes resemble those associated with coronary artery occlusion. Gore (54) pointed out that although any coronary artery or vein lying in the path of force transmitted from blunt impact to the precordium is theoretically capable of being bruised or lacerated, the vascular injury incurred in such circumstances is ordinarily a relatively insignificant feature of total trauma. The larger epicardial arteries and veins are characteristically less vulnerable to injury by crushing or distorting injuries than are the tissues around them. A

blunt injury to the chest of great enough intensity to cause bruising or laceration of a coronary vessel will almost invariably produce concomitant myocardial damage of greater importance. Gore (54) stated that in special circumstances the effects of trauma may be so sharply localized that vascular contusion is the most significant feature of injury, and although it might seem plausible that coronary arteries and veins were equally susceptible to such trauma, the significance of venous lesions incurred in this manner is negligible.

Gore (54) pointed out that intense force is necessary to produce an injury of a vessel which will result in formation of a thrombus, and that a thrombus formation in the lumen of a damaged artery rarely results in occlusion, and is characteristically confined to the mural defect where it organizes as part of the reparative reaction. It will be pointed out in the section on animal experimentation that it is extremely difficult to produce traumatic coronary artery occlusion in animals, and this fact justifies critical appraisal of the evidence when it is thought that such a thrombus has occurred in man.

Gore (54) states:

> The facts (1) that active persons and particularly those engaged in physically arduous occupations frequently sustain thoracic impacts, and (2) that atherosclerotic heart disease is one of the most common causes of disability and death among the adult population, make it inevitable that many persons develop coronary thrombosis after sustaining some form of thoracic trauma. In most instances the sequence is fortuitous and not indicative of a cause-effect relationship. We cannot exclude the possibility that blunt impact may in certain special circumstances produce, in an already diseased artery, sufficient localized injury to percipitate thrombosis. If coronary thrombosis is known to have developed within a few days after a severe precordial impact, and if the site of thrombosis corresponds to the path of the transmitted force, the possibility of a cause-effect relationship needs to be entertained. If evidence of myocardial contusion or laceration in the vicinity of the vascular lesion is found at post mortem examination of such

> an individual, the possibility that trauma contributed to the development of thrombosis would be reasonably certain.

Gore (54) described one case in which the victim died 5 days after sustaining a crushing injury to the chest. Autopsy revealed a transverse fracture of the body of the sternum, hemorrhage throughout the anterior mediastinum, laceration of the parietal pericardium, contusion of the epicardium and myocardium over the anterior portion of the interventricular septum, recent thrombosis of the atherosclerotic proximal segment of the descending ramus of the left coronary artery and recent infarction of the tip of the left ventricle. He stated that it was not clear whether the thrombus had occurred because of direct vascular injury or because of the state of the systemic circulatory stasis and shock which followed the injury; there was no reason to doubt a cause-effect relationship between thrombosis and trauma in this instance.

Gore (54) also concluded that in practically every case of coronary artery occlusion or coronary thrombosis, the thrombus develops on the basis of preexisting atherosclerosis, and he stated:

> Instances of traumatic coronary thrombosis with fresh myocardial infarction are cited by Monckenberg and Bean who encountered three instances of traumatic myocardial infarction among 9629 consecutive autopsies. One of these concerned a patient who fell eight feet from a ladder, sustaining fractures of the ribs of the left side. He died 10 weeks later and at autopsy a recent infarct with beginning aneurysm was present. H. Levy reported an example of traumatic coronary thrombosis with a myocardial infarct in a woman of 49 who sustained a contusion of the chest wall in an automobile accident and died 13 days later. Autopsy revealed hemorrhage beneath the intima, rupture of the intimal lining and thrombosis in the lumen of the anterior descending branch of the left coronary artery, together with a large myocardial infarct involving the ventral half of the entire apex. The microscopic appearance of the thrombus and the infarct were consistent with changes which could occur in the interval between the accident and death. Friedberg stated that in cardiac wounds the coronary arteries are commonly involved, particularly the anterior descending branch of the left coronary (artery). In

the application of nonpenetrating blunt force to the chest wall the ventral position of the right coronary artery is said to render this vessel more vulnerable to injury than the left artery.

Valvular Lesions

Barber (2) pointed out that most valvular lesions resulting from trauma have a well defined history of immediate distress and have probably shown some unusual physical signs. Barber (2) quoted Wilks who described the case of a young man who died after falling from a height, and striking his left side on a large stone. Autopsy revealed that the immediate cause of death was due to abdominal injuries; however, there was a cardiac lesion, rupture of the posterior cusp of the aortic valve, which was split from the free edge to the base. Gibson (62) reported an immediate death from mitral valve rupture resulting from a kick to the chest by a horse. Kemp (63) described traumatic rupture of the aorta in a 46-year old man who was struck in the chest by a portion of a stone fly-wheel. Autopsy revealed hemopericardium due to a transverse tear just anterior to the aortic valves. Kissane, Fidler and Koons (64) recorded the rupture of the aortic valves in the victim of an explosion.

Most traumatic valvular lesions show a well defined downward course from time of injury until death (2).

Barber and Osborn (65) reported a case of mitral valve lesion resulting from heart contusion in a miner injured in an explosion. The man was bedfast for five years following the accident with a mitral systolic bruit as the chief sign of a disorganized mitral valve. Death from pneumonia occurred 22 years after the original incident and an autopsy revealed that this lesion had healed up to mitral stenosis. There was

scar tissue throughout the left ventricular wall, continuous with the valve. Warburg (22) reported a crushing injury to the chest of a 20-year old man, who demonstrated obvious evidence of valvular lesion; however, a presystolic mitral bruit was not evidenced until three years after the accident. Animal experimentation has confirmed that trauma may cause bruising at the base of the valves, and it is possible that this might lead to some degree of fusing of the cusps (2).

Gore (54) stated that the cardiac valves, their chordae tendineae, and their papillary muscle may be lacerated by impact, by hydrostatic forces transmitted from sites of trauma elsewhere in the body, or by the strain of over-exertion. It has been pointed out that:

> The largest series of traumatic rupture of normal valves is that reported by Adam in 1927, and includes 16 cases, of these, seven were aortic, five of the mitral, one of the aortic and mitral, two of the pulmonic, and one of the tricuspid. White and Glendy have stressed the importance of being most critical of the evidence purporting to establish that a diseased valve has become insufficient because of trauma. Unless it can be shown that the valvular insufficiency developed after the trauma and not before, and that the trauma or stress was of such a nature as to be consistent with the production of a disruptive force against the valves, the sequence in question should be regarded as unproved. If traumatic rupture of an atrioventricular valve is suspected, the heart should be opened with great care so as to avoid cutting the chordae tendeneae (66).

Animal Experimentation

There is ample evidence from animal experimentation to support the conclusion that the force of a blunt impact to the chest may cause a wide variety of nonfatal or fatal disturbances in cardiac function without visible evidence of injury. Kulbs (67) began the original experimental work with dogs subjected to blunt thoracic trauma. Barber (2) quoted Kulbs and Strauss who recorded injury of the heart in dogs, with the skin

and the thorax intact, and in some instances observed bradycardia.

Posttraumatic Dysrhythmia

The mechanism of traumatic dysrhythmia is obscure. Moritz (3) quoted Schlomka and Schmitz who concluded from animal experimentation that the cardiac disturbances caused by commotion are probably due to reflex coronary vasoconstriction and myocardial ischemia. Moritz (3) also quoted Kastert who reported the findings of focal areas of necrosis and myocardial degeneration in relation to the coronary termini in animals that had survived the immediate effects of nondisruptive cardiac impact. In experiments on dogs by Schlomka and Schmitz (68) there were posttraumatic changes in the QRS and T waves of the electrocardiograms, and bundle branch blocks; the changes were similar to those which result from coronary insufficiency, and were accompanied by a drop in arterial blood pressure, a rise in venous blood pressure, and dilatation of the right heart. Bright and Beck (7) exposed the heart in 25 dogs and subjected it to bruising by a metal hammer. The hearts were exposed under anesthesia before the trauma was applied and the actual bruising was observed. There was often an immediate and extreme rise in pulse rate, with falling arterial pressure, and rising venous pressure. They noted cardiac dilatation, tachycardia, and occasionally bradycardia. There were abnormal electrocardiograms indicating myocardial lesions, and in some cases hemorrhage was found in the interventricular septum. One dog died shortly after trauma from myocardial failure, and two died from ventricular fibrillation. In the animals which survived, cardiac dilatation often persisted for several weeks, and the electrocardiographic changes were similar to those observed by Schlomka and Schmitz (68). The disappearance

of these changes tended to parallel the reduction in size of the cardiac silhouette. When the animals were sacrificed 2 or 3 months afterwards, the myocardial damage was repaired and pericardial adhesions were noted. The authors were surprised to find that extensive bruising was compatible with recovery.

In a series of dogs in which the heart was exposed and subjected to nonfatal blunt impact, Moritz and Atkins (69) found that structural evidence of cardiac injury was absent in three of five animals that developed posttraumatic extrasystoles, in one of two that developed bradycardia, in two of six that developed tachycardia and in two of seven that developed ventricular fibrillation.

Fasola, Baker and Hitchcock (44) quoted Schlomka who experimentally produced myocardial hemorrhage in dogs heart and observed a fall of the arterial blood pressure with a simultaneous rise in the venous blood pressure. He found that acute dilatation of the heart was most common. There were temporary abnormalities present in the electrocardiographic tracings, which he thought might be due to spasm of the coronary arteries. Posttraumatic dysrhythmias including extrasystoles, bradycardia, and tachycardia were also noted. Arenberg (4) in reviewing the work of Bright and Beck concluded that the electrocardiographic changes they observed were not unlike those seen in man with various stages and degrees of myocardial damage. Some of the changes disappeared after a month or so, and others persisted for some time following trauma. He concluded that healing of a nonfatal cardiac injury is the rule, and on the basis of experimental and clinical observations, it was felt that the vast majority of nonpenetrating wounds of the heart are not recognized clinically or do

not receive the correct diagnosis. Gore (54) stated that many if not most of the functional disturbances of the heart that have been observed in animals following blunt impact to the precordium may follow cardiac trauma in man.

Injury of the Coronary Arteries and Veins

The findings of posttraumatic dysrhythmia reported by Moritz and Atkins (69) have been discussed; however, this series of experiments also revealed that trauma to the exposed heart in the area over the descending ramus of the left coronary artery was not without consequence. The only instance in which recognizable vascular injury was sustained were in those experiments in which gross lacerations of the myocardium occurred. Severe myocardial bruising was produced without appreciable damage to the coronary vessels lying directly beneath the site of impact. In unpublished experiments reported by Gore (54), Moritz observed that it was necessary to crush an artery between the jaws of a clamp in order to produce a localized vascular injury of sufficient intensity to result in thrombosis. Even in such circumstances, the production of sufficient trauma to predispose to thrombosis is likely to result in the formation of a false aneurysm as a result of the escape of blood into the wall of the vessel.

Randles, Gorham and Dreskback (70) in experimental work on changes in the RS-T component of the ECG produced by experimental rupture of the auricle of dog's heart and by pericardial injection, observed an electrocardiographic picture which was suggestive of coronary artery occlusion and pericarditis.

Myocardial Damage

It has been observed both in persons and in experimental animals that a blunt injury of the heart may lead to widely disseminated myocardial hemorrhages without visible laceration. These are apparently the result of minute focal lacerations of muscle and probably result from an impact being delivered while the ventricles are filled with blood (54).

Both Munck and Warburg as reported by Gore (54) have stressed the frequency of myocardial hemorrhage in humans. The experimental findings of Moritz and Atkins (69) suggest that the hydrostatic forces incident to sudden compression of the heart between the sternum and the ribs anteriorly and the vertebral column posteriorly is a frequent cause of myocardial rupture. Lateral displacement of the heart by an obliquely directed force may lacerate the pericardium without other damage to the heart or may tear the wall of the left atruim at the ostia of the pulmonary veins. In cardiac laceration incident to falls from a height, partial or complete circumferential laceration of the aorta immediately above the aortic valves is sometimes encountered.

Zuckerman, Krohn, and Witteridge (71) carried out experiments on animals exposed to compressive blasts. Although they were primarily interested in the effects on the pulmonary system they found some evidence of myocardial injury and some abnormal electrocardiographic findings which were similar to those found in clinical examples of myocardial contusion. Kissane, Fidler and Koons (34) carried out experiments on dogs which produced similar results to those which were observed by Bright and Beck (7) although they noted one ruptured valve and one animal that died from ventricular fibrillation. Joeffe (14) using dogs, has shown

that congestion of the lungs, interpreted as congestive heart failure, is the primary pathological result of rapid deceleration into lap type seat belts, probably resulting from the effects of hydrostatic forces of the cardiovascular system. He believes that higher deceleration forces than those produced in his work, might result in permanent deformation of the myocardium. Fasola, Baker and Hitchcock (44) experimentally subjected dogs to varying degrees of chest injury. The results were recorded on electrocardiographs and collected from gross and microscopic autopsy findings. There was some uncertainty as to how their data might be applied to humans and further attempts were planned for the collection of industrial and automotive reports regarding chest trauma. The results of this phase of the study are not known.

Electrocardiographic and Physical Changes Associated with Blunt Thoracic Trauma

Crushing injuries and direct blows to the precordium have caused cardiac rupture with hemopericardium, myocardial contusion and rupture of a valve or of the chordae tendineae (2, 7, 41, 54, 68). Myers (72) stated that when blunt thoracic trauma has caused extensive myocardial contusion or when a coronary artery has been severed or surgically ligated, abnormal Q waves, typical of those found in myocardial infarction are demonstrable, and the resulting pathological lesion may heal to leave a cardiac aneurysm. Death from the acute stage may result from ventricular fibrillation or cardiac arrest. Arterial injuries resulting from indirect blows may be further complicated by atrial fibrillation or flutter, both of which are generally transient. Rupture of the aortic valve by nonpenetrating trauma is manifested by the sudden appearance of a loud sea-gull diastolic murmur

over the entire precordium, accompanied by peripheral signs of aortic regurgitation and the rapid development of congestive heart failure. Mitral valve or chordae tendineae rupture cause a loud systolic murmur and thrill, which is maximal at the apex and transmitted over the entire precordium, generally followed by congestive heart failure.

Randles, Gorham and Dreskback (70) examined changes in the RS-T component of the electrocardiogram produced by experimentally rupturing the atrium of a dogs heart and by pericardial injection. Their electrocardiographic findings were suggestive of coronary artery occlusion and pericarditis. The electrocardiograms from the experiments of Bright and Beck (7) showed a variety of alterations from the normal; the most frequent variation was the production of large T waves and alterations in the Q wave. Frequently, there was a high take-off of the T wave and in some cases the T wave was inverted. Slurring and notching the QRS complex was also observed. These deviations from the normal electrocardiograms, in great part, had disappeared after about 1 month, but some of the changes persisted for a longer time. It is interesting to note that the electrocardiogram obtained in the experiments in which blood was injected into the interventricular septum was somewhat similar to the electrocardiogram obtained in which the myocardium was bruised. They pointed out that this change, together with the fact that most of the electrocardiographic changes disappeared within a few weeks, would indicate that these alterations are due largely to hemorrhage. The changes in the electrocardiograms were somewhat similar to the electrocardiographic changes accompanying tamponade or arterial occlusion with myocardial degeneration due either to arterial or arteriolar sclerosis and

are not unlike those seen in man with various stages and degrees of myocardial involvement (72). Kissane (73) in his correlated studies of experimental cardiac injury in 19 dogs and of cardiac damage in 14 humans confirmed the fact that severe injury to the heart can take place in thoracic trauma without fracture. Electrocardiographic findings demonstrated changes in the T wave and ST segments of the QRST complex, some of which were transient and some of which persisted from 12 to 18 months.

Barber (37) published the records of 75 unselected accident cases. Electrocardiograms were taken within 48 hours following injury, and 20 patients with abnormal electrocardiograms were discovered. In one patient, partial heart block persisted permanently; however, in the other patients, the electrocardiograms returned to normal in a relatively short time. Barber accepted this as evidence that the abnormalities were the result of trauma. These temporary abnormalities were found most frequently 1 or 2 days after injury. On several occasions there was a normal tracing a few hours after the accident, whereas 24 or 48 hours later, abnormalities appeared. This was followed by a normal tracing, sometimes a few days after the second tracing and in some cases in the second or third week. In one or two elderly men it was 4 or 5 weeks before an inverted T wave from lead I returned to an upright position. Premature contractions were met with, but not frequently and no attempt was made to assess their significance. The T wave in lead III was inverted temporarily on one or two occasions, but it seemed to Barber, that this was possibly the result of a change in the height of the diaphragm. None of the patients in this series had pneumothorax which was significant because, as Master (74) pointed out, this condition may temporarily cause

changes in the ECG.

Barber (37) carefully pointed out that the trauma itself was of such a varying type and degree that a much larger series of patients would be required before any statements could be made regarding age specificity or liability to particular accidents which might cause heart lesions. He stated that the series seemed to confirm the belief that an ECG should be taken soon after an accident and repeated at regular intervals thereafter.

It can be concluded that changes in the heart, resulting from nonpenetrating or blunt trauma may not occur immediately. Arenberg (4) believed that there was no doubt that many cases of myocardial contusion or damage to the heart in nonpenetrating chest trauma are missed and not diagnosed. Since physical signs of cardiac contusion or of myocardial damage are often absent if the pericardium is not involved, the condition may not be recognized unless repeated ECG's are made. The symptoms of pain, he continues, particularly in respiration are usually ascribed to muscular contusion or to "traumatic pleurisy". Moreover, definite symptoms of anginal syndrome or of myocardial insufficiency may develop or become apparent later, after recovery from the initial chest trauma had taken place.

Fasola, Baker and Hitchcock (44) observed electrocardiographic changes in dogs which were rapidly decelerated into lap type abdominal seat belts, and concluded that the electrocardiogram provides a useful means of diagnosing cardiac injury if frequent tracings are taken following trauma.

Many of the electrocardiographic changes which they observed

have definitely been associated with coronary artery occlusion and pericarditis (10, 34, 37, 43, 75). They noted slurring and temporary changes in direction of the "R" wave, voltage changes in the QRS component, and axis deviation. The results of the ECG's in this series of animal experiments were summarized as to rhythm, P-R, QRS, QRST intervals, heart rate, and individual measurements in the amplitude of the P, Q, R, S, T waves and the level of the ST segment in leads I, II and III. On the basis of their results they concluded that inversion of the T wave with simultaneous elevation of the ST segment is evidence of cardiac trauma. Other bizzare changes including enlargement of the Q wave and changes in the contour of the QRS complex occurred, but since the records could not be accurately described, no attempt was made to correlate them with any specific heart damage.

Moritz (5) summarized the various functional disturbances which may result from penetrating and nonpenetrating thoracic trauma (Figure 1).

Gore (54) is somewhat skeptical of the relationship between nonpenetrating thoracic injury and cardiac damage. He believed that no clinical consequence of a nonpenetrating cardiac injury is pathognomonic of trauma. He stated that although cardiac trauma may lead to the various disturbances indicated in Figure 1, the mere occurrence of such a disturbance following a thoracic injury does not, of itself, constitute proof that it was caused by the injury. He maintains that a functional disturbance of the heart that has been caused by trauma will invariably manifest itself almost immediately, and the longer the interval between the occurrence of an injury and the subsequent development of signs or symptoms of cardiac disturbance, the less is the likelihood that there is a cause effect relationship between them.

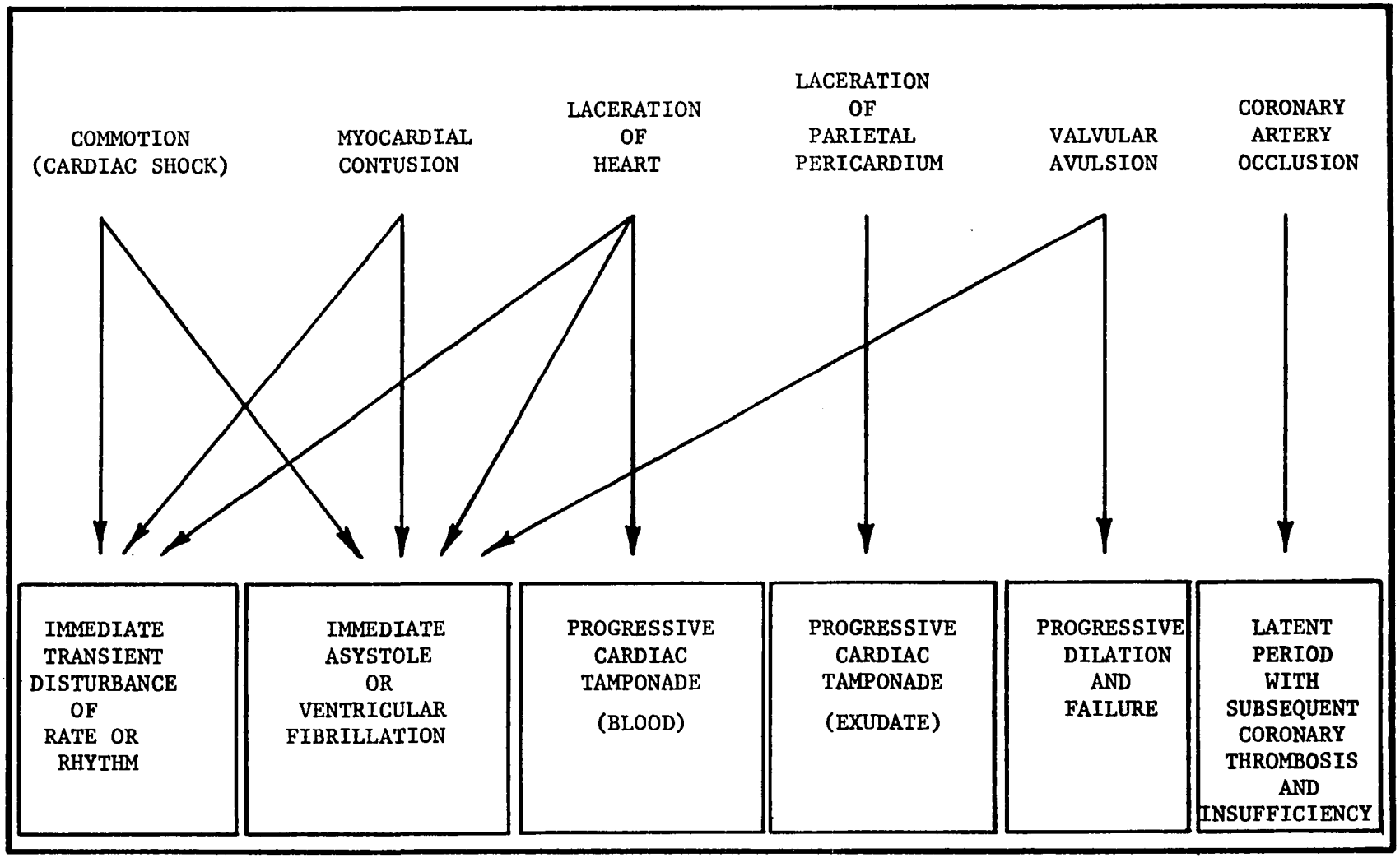

Figure 1--Direct injury of the heart by blunt and nonpenetrating injuries to the thorax [from Moritz (15)].

CHAPTER II

PURPOSE AND SCOPE

The literature indicates that the etiology and pathology of heart disease is extremely diverse; a single pathological condition may result from numerous causative mechanisms or combinations of these mechanisms.

The purpose of this study was to determine whether electrocardiographic evaluation of drivers in motor vehicle accidents would reveal evidence of cardiac damage.

Electrocardiography was selected as the experimental procedure because variations from normal electrical activity of the heart have been categorized and correlated with specific pathological states.

Numerous authors have been cited who reported cases of cardiac damage resulting from a wide variety of traumatic experiences. It seemed reasonable, therefore, that a motor vehicle driver might sustain cardiac damage in a collision.

A methodology was designed which provided the opportunity of investigating the frequency of abnormal electrocardiographic findings in recent "driver - victims" as well as drivers who had been involved in collisions during a period of time extending back 5 years prior to the studys inception. In addition, provisions were made to investigate the frequency of electrocardiographic abnormalities in a selected

"accident-free" sample and thereby determine whether drivers who had accident experience demonstrated a significantly different frequency of electrocardiographic abnormalities than did their matched counterparts.

CHAPTER III

PROCEDURES AND EQUIPMENT

Subjects

The study sample was comprised of three groups, to be designated hereafter as Group I, Group II and Group III.

Selection of Group I subjects was based upon evaluation of motor vehicle accident reports collected by investigators from the Protection and Survival Laboratory of the Civil Aeromedical Institute (CAMI) during a 5 year period prior to the study.

Subjects selected for Group I were vehicle drivers that the reports indicated had likely sustained blunt thoracic trauma.

Also, to be included in Group I, the subject must have been:

(A) Examined and/or treated in a hospital emergency room or a physicians office for some type of injury sustained in the motor vehicle accident.

(B) Free from any clinical history of heart disease.

(C) Free from past history of blunt thoracic trauma.

(D) Willing to participate in the study.

Selection of Group II (a control population) involved matching Group II subjects with Group I subjects in terms of the following variables:

(A) Age

(B) Sex

(C) Race

(D) Height

(E) Weight

Also to be included in Group II, subjects must have:

(A) No previous involvement, as a driver, in a motor vehicle accident involving personal injury.

(B) No clinical history of heart disease.

(C) No previous history of blunt thoracic trauma.

(D) Displayed willingness to participate in the study.

Subjects for Group III were drivers who had been involved in motor vehicle accidents and were taken into the emergency room facilities of the Baptist Memorial Hospital, Oklahoma City, Oklahoma during a 43-day testing period between December 19, 1969 and January 31, 1970.

No further criteria for selection was required of Group III subjects, and apparent thoracic injury was not necessary in order that the driver be included in the study sample. The selection criteria were so established in hopes of demonstrating electrocardiographic abnormalities in the absence of clinical symptoms, or thoracic fractures involving the ribs and/or sternum.

Sample Structure

In the analysis of cardiac damage resulting from nonpenetrating steering wheel impact, the three groups comprising the study sample were characterized as follows:

Table 1 Group I - Drivers Sex and Year of Accident

TABLE 1

GROUP I - SEX OF DRIVERS AND YEAR OF ACCIDENT

Year of Accident	Number of Drivers	Male	Female
1965	2	1	1
1966	8	6	2
1967	3	2	1
1968	13	8	5
1969	4	1	3
Totals	30	18	12

Table 2 - Sex Composition of Groups I, II and III.

Table 3 - Minimum, Maximum and Mean Values for Age, Height and Body Weight - Groups I and II.

Height and weight measurements were obtained on each subject in Group I and Group II, however it was not possible to obtain similar data on subjects in Group III.

Table 4 - Minimum, Maximum and Mean Age Values for Group III.

Measurements

Arrangements were made with the Branch Chief of the Employee Health Clinic at CAMI to utilize the facility for obtaining the electrocardiograms on members of Groups I and II.

All electrocardiograms on Group I and II subjects were taken by the author.

In numerous instances Group I subjects were unable to come into the clinic and arrangements were made to obtain the tracings at the subjects' home.

All electrocardiograms on Group I and II subjects were taken on the Sanborn Visette - Model 300 electrocardiograph. This instrument is designed for a 115 volt - 60 cycle power line.

The Visette is basically a recording vacuum tube voltmeter for making a permanent record of heart voltages, in rectangular coordinates. Recording sensitivity is 1 millivolt input per 10 divisions of deflection on the recording.

Vertical measurements were in true millimeters (with every fifth millimeter line accentuated) for a total channel width of 35 millimeters. Time axis deviations were always in true millimeters, with every fifth

TABLE 2

SEX COMPOSITION OF GROUPS I, II AND III

Group	Male	Female	Totals
I	18	12	30
II	18	12	30
III	41	13	54
Totals	77	37	114

TABLE 3

MINIMUM, MAXIMUM AND MEAN VALUES FOR AGE, HEIGHT AND WEIGHT - GROUPS I AND II

Group	N	$\bar{X}$ - Age	$\bar{X}$ - Height	$\bar{X}$ - Weight
WHITE				
Males - Group I	16	20 - 67 34.93	64" - 72" 70.32"	150 - 190 171.343
Males - Group II	16	20 - 65 35.50	68" - 72" 70.17"	138 - 185 170.31
Females - Group I	11	19 - 64 33.72	60" - 70" 64.52"	97 - 169 130.45
Females - Group II	11	21 - 65 33.80	62" - 66.5" 64.50"	102 - 165 130.45
NEGRO				
Males - Group I	2	21 - 37 28.50	67" - 68" 67.50"	135 - 180 157.5
Males - Group II	2	22 - 38 30.00	68" - 70" 69.00"	138 - 179 158.5
Females - Group I	1	N = 1 16.00	N = 1 60.00"	N = 1 75.0
Females - Group II	1	N = 1 16.00	N = 1 62.00	N = 1 90.0

TABLE 4

MINIMUM, MAXIMUM AND MEAN AGES FOR GROUP III SUBJECTS

Group III	N	$\bar{X}$ - Age
WHITE		
Male	40	11 - 75 29.50
Female	13	16 - 70 34.15
NEGRO		
Male	1	N = 1 28.00
Female	--	-----

millimeter line accentuated for easier reading of the final recording at a paper speed of 25 millimeters per second.

Twelve lead electrocardiograms were taken on Group I and Group II subjects. The twelve lead electrocardiograms includes the "bipolar limb leads" designated as I, II and III. Lead I is the pairing of the right arm and left arm, Lead II is the pairing of the right arm and left leg, and Lead III is the pairing of the left arm and left leg.

Additional leads are the "unipolar limb leads" designated as aVR, aVF and aVL, which are sometimes referred to as "augmented" unipolar limb leads.

A unipolar limb lead, as contrasted with the bipolar limb lead is one which represents the heart voltage present at one particular spot on the body with respect to a nonfluctuating reference point. This reference point is secured by connecting the limb electrodes together through three equal resistances of 5000 ohms or more; the junction of the three, is the reference point, or central terminal, which is embodied in the lead selector of the instrument.

The electrodes placed on the body are then paired, in turn, with this reference point or central terminal, to secure the unipolar leads which, as previously noted, represent the actual voltages present at these single points on the body.

Upper extremity limb leads were taken with electrode placement on the inner aspect of the forearms, slightly superior to the area of the radial pulse.

Lower extremity limb leads were taken with electrode placement on the inner aspect of the lower leg, slightly superior to the calcaneus

bone and slightly inferior to the distal end of the gastrocnemius muscle. Six precordial or chest leads were taken with a suction-cup type electrode. The precordial electrode placement included the following positions:

A. Position 1 - The fourth intercostal space, at the right border of the sternum.

B. Position 2 - Same intercostal interspace, at the left sternal border.

C. Position 3 - Midway between positions 2 and 4.

D. Position 4 - Fifth intercostal interspace, at left mid-clavicular line.

E. Position 5 - Same level as 4, in anterior axillary line.

F. Position 6 - Same level as 4 and 5, in midaxillary line.

Figure 2 illustrates the electrode position placement for Group I and Group II precordial leads.

Electrocardiograms obtained from 30 selected subjects comprising Group I and 30 matched control subjects comprising Group II were coded as to group and subject identity and submitted to Dr. Charles M. Brake, Branch Chief of the Employee Health Clinic for evaluation and diagnosis.

Electrocardiograms on Group III subjects were taken in the emergency room of the Baptist Memorial Hospital, Oklahoma City, Oklahoma by the author or a selected electrocardiographic technician, soon after the subject reached the hospital. All vehicle drivers brought into the BMH emergency room during the specified period of the study were included in Group III. No criteria for severity of physical injury was established as a prerequisite for becoming a study population member, in an attempt to assess potential electrocardiographic changes in any vehicle driver

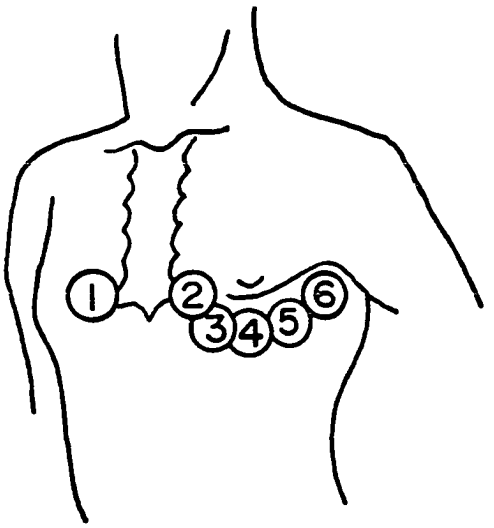

Figure 2--Electrode positions - Groups I and II - Precordial Leads.

regardless of the nature and extent of physical trauma.

Single tracings were obtained on all subjects, and serial electrocardiograms were obtained on subjects who were admitted into Baptist Memorial Hospital, and on those subjects who were not admitted, but agreed to return at designated 24- and 48-hour intervals for comparative tracings.

A Cambridge Electrocardiograph was utilized for all Group III tracings. This instrument is quite similar to the Sanborn Visette - 300 which was used for Groups I and II.

It is a transistorized unit incorporating a voltmeter which permanently records heart voltages in rectangular coordinates. Recording sensitivity is 1 millivolt input per 10 divisions of deflection on the recording. Vertical measurements were also in true millimeters (with every fifth millimeter line accentuated) for a total channel width of 35 millimeters. As with the Sanborn Visette, time axis deviations were always in true millimeters with every fifth millimeter line accentuated for easier reading of the final tracing at a paper speed of 50 millimeters per second.

The electrocardiograms on Group III subjects consisted of 17 leads, five more diagnostic leads than were utilized for Groups I and II. These additional leads included:

A. Position 7 - v4R - Fifth interspace at right midclavicular line.

B. Position 8 - V7 - Same level as V4 and V5 in left posterior axillary line.

C. Position 9 - V8 - Same level as V7 in left midscapular line.

D. Position 10 - V9 - Same level as V7 in posterior midline.

E. Position 11 - VB - Apex of triangle formed by VB - V7 and V9 at level of fourth intercostal space on back.

Figure 3 illustrates the additional electrode position placements for Group III.

Electrocardiograms on Group III subjects were obtained on an "around-the-clock" basis, with the author answering all emergency room alerts during those hours when a selected electrocardiographic technician was not available.

The electrocardiograms were submitted to Dr. John J. Donnell, a cardiologist, and the Director of the Baptist Memorial Hospital Heart Station, and to Dr. Merle C. Carter, a Resident in Internal Medicine at BMH for evaluation and diagnosis.

Evaluation and diagnosis of electrocardiograms for subjects from all three groups appears in Appendix 1.

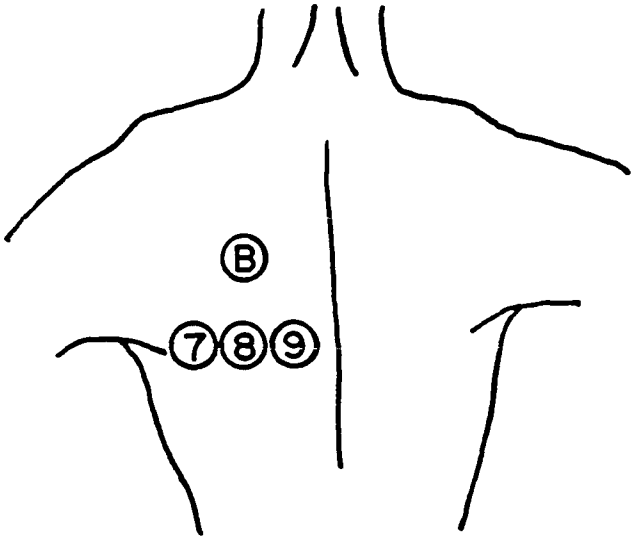

Figure 3--Additional electrode positions - Group III.

CHAPTER IV

RESULTS

One hundred fourteen electrocardiograms were obtained and analyzed in this study.

Eighty-four subjects had been drivers in motor vehicle accidents. Thirty of the above had been involved in an accident sometime during a 5-year period prior to the study. Fifty-four subjects had been drivers in a motor vehicle accident within minutes of their electrocardiograms.

Analysis of electrocardiograms in Group I revealed that 50 per cent of these drivers demonstrated some electrocardiographic abnormality of varying degrees of severity (Table 5).

An analysis of a matched control sample (Group II) revealed that only 23 per cent of these subjects demonstrated any electrocardiographic abnormalities (Table 5).

Electrocardiograms of Group III subjects revealed that 65 per cent demonstrated some electrocardiographic abnormalities soon after a motor vehicle accident, and only 35 per cent of these subjects were found to have normal heart tracings (Table 6).

Comparison of the percentage of abnormal and normal electrocardiographic findings between Group I and Group II indicated that twice as many subjects who had been drivers in motor vehicle accidents during a 5-year period prior to the study demonstrated electrocardiographic

TABLE 5

NUMBER OF SUBJECTS WITH ELECTROCARDIOGRAPHIC ABNORMALITIES - GROUPS I AND II

	Subjects	Subjects with Abnormal ECG Findings	Subjects with Normal ECG Findings	Percentage of Subjects with Abnormal Findings
Group I	30	15	15	50
Group II	30	7	23	23

TABLE 6

NUMBER OF SUBJECTS WITH ELECTROCARDIOGRAPHIC ABNORMALITIES - GROUP III

	Subjects	Subjects with Abnormal ECG Findings	Subjects with Normal ECG Findings	Percentage of Subjects with Abnormal Findings
Group III	54	35	19	64.8

abnormalities than did a matched sample from Group II.

Positive findings in Group III (65 per cent) were almost twice as great as the percentage of negative findings (35 per cent).

In both instances, positive electrocardiographic abnormalities are noted with approximately twice the frequency of negative findings.

A Chi-square test was performed on the number of abnormal and normal tracings in Groups I and II. The Chi-square value was determined to be 4.593 which is significant at the 0.05 level with one degree of freedom. On the basis of this statistical analysis it was determined that there was a significant difference between the number of subjects with electrocardiographic abnormalities in Group I and the matched sample Group II.

A more detailed analysis of Group I subjects revealed that 16 abnormal conditions were observed in those tracings which demonstrated positive findings.

Sinus arrhythmia was diagnosed most frequently, occurring in three subjects, one of whom, a 21-year old white male was also found to have right axis deviation. A first degree A-V block with possible left ventricular hypertrophy was observed in a 37-year old Negro male. Only one other Group I subject was diagnosed as possibly having left ventricular hypertrophy.

Left axis deviation was revealed in a 51-year old white male who also demonstrated nonspecific T wave abnormalities; left axis deviation was also found in a 42-year old white female who was further diagnosed as having both sinus bradycardia, and poor R wave conduction across the precordium.

A 20-year old white female demonstrated changes which were compatible with an embolic phenomena, a condition which was clinically diagnosed during her hospital tenure.

Possible subepicardial injury of the inferoseptal and posterolateral heart surfaces were noted in a 20-year old white male, who, in 1968, struck a bridge abutment.

Atrial enlargement was observed in a 38-year old white female and the Lown-Ganong-Levine (LGL) syndrome was observed in a 47-year old white female who had been injured 9 months prior to the tracing.

A-V nodal rhythm and premature nodal contractions were found in two young white females, ages 20 and 19 years respectively.

Inverted to diphasic T waves were observed in a poorly nourished negro female, however, it was felt that this could be normal for this subject, or perhaps the retention of a juvenile pattern.

Sinus tachycardia was observed in only one subject, a 29-year old white male (Table 7).

As mentioned previously, approximately 23 per cent of the Group II subjects demonstrated electrocardiographic abnormalities.

Sinus bradycardia was observed in two males. Sinus arrhythmia was noted in a 28-year old white male and in a 38-year old negro male.

A 35-year old white female demonstrated "U" waves in precordial leads VI through V4 as well as showing electrocardiographic evidence of sinus tachycardia.

The most significant finding in the control group was that of an old anteroseptal myocardial infarction in a 65-year old white female, although the reader did not believe that the tracing was diagnostic.

TABLE 7

GROUP I - ELECTROCARDIOGRAPHIC ABNORMALITIES

Abnormality	Number of Subjects with Abnormality
Sinus Arrhythmia	3
Sinus Bradycardia	1
Sinus Tachycardia	1
Left Axis Deviation	1
Right Axis Deviation	1
Possible Left Ventricular Hypertrophy	2
First Degree A-V Block	1
Nonspecific T Wave Abnormalities	1
Atrial Enlargement	1
Lown-Ginong-Levin Syndrome	1
A-V Nodal Rhythm	1
Poor R Wave Conduction Across Precordium	1
Inverted To Diphasic T Waves	1
Premature Nodal Contractions	1
Changes Compatible with Embolic Phenomena	1
Possible Subepicardial Injury	1

NOTE: Some subjects demonstrated more than one electrocardiographic abnormality, therefore total abnormalities will exceed the total number of subjects.

A 65-year old white male was found to have left axis deviation and 0.5 to 0.75 millimeter S-T segment depression in leads II, aVF, V4, V5, and V6 and these findings are compatible with subendocardial ischemia (Table 8).

Table 9 indicates the electrocardiographic abnormality observed in Group I and II subjects and lists the number of subjects demonstrating the particular electrocardiographic finding.

A total of 39 single tracings only, were obtained on Group III subjects, which included 17 subjects with normal findings and 22 subjects with abnormal findings (Table 10).

Primary nonspecific T wave abnormalities were noted in 7 subjects. Three subjects with these T wave abnormalities also demonstrated additional findings which included possible subepicardial injury in a 22-year old white male, atrial fibrillation with an average ventricular rate of 45, in a 56-year old white male, and sinus tachycardia in a 19-year old white male.

Sinus tachycardia was observed in 4 other subjects, and of these 4, one subject demonstrated ventricular extrasystoles.

Ventricular extrasystoles, alone, were noted in a 75-year old white male.

Incomplete right bundle branch block was observed in five subjects. Two of the five also showed indications of left ventricular hypertrophy. Left ventricular hypertrophy alone, was found in two other subjects, and right ventricular hypertrophy, the more serious condition, was observed in a 20-year old white male.

A 16-year old white male was observed to have a first degree

TABLE 8

GROUP II - ELECTROCARDIOGRAPHIC ABNORMALITIES

Abnormality	Number of Subjects with Abnormality
Sinus Arrhythmia	2
Sinus Bradycardia	2
Sinus Tachycardia	1
Myocardial Infarction (Old)	1
Subendocardial Ischemia	1
Left Axis Deviation	1

NOTE: Some subjects demonstrated more than one electrocardiographic abnormality, therefore total abnormalities will exceed the total number of subjects.

TABLE 9

CLASSIFICATION OF ECG FINDINGS - GROUPS I AND II

Condition	Number of Subjects with Abnormality GROUP I	Number of Subjects with Abnormality GROUP II
Sinus Arrhythmia	3	2
Sinus Bradycardia	1	2
Sinus Tachycardia	1	1
Myocardial Infarction	0	1
Left Axis Deviation	1	0
Right Axis Deviation	1	0
Possible Left Ventricular Hypertrophy	2	0
First Degree A-V Block	1	0
Nonspecific T Wave Abnormalities	1	0
Atrial Enlargement	1	0
Lown-Ginong-Levin Syndrome (LGL)	1	0
A-V Nodal Rhythm	1	0
Poor R Wave Conduction Across Precordium	1	0
Inverted to Diphasic T Waves	1	0
Premature Nodal Contractions	1	0
Changes Compatible with Embolic Phenomena	1	0
Possible Subepicardial Injury*	1	0
Subendocardial Ischemia	0	1

*Inferolateral and posterolateral surfaces.

TABLE 10

GROUP III - ELECTROCARDIOGRAPHIC ABNORMALITIES
SINGLE TRACINGS

Abnormality	Number of Subjects with Abnormality
Nonspecific T Wave Abnormalities	7
Right Bundle Branch Block (Incomplete)	5
Sinus Tachycardia	5
Left Ventricular Hypertrophy	4
Ventricular Extrasystoles	2
Right Ventricular Hypertrophy	1
S-T Segment Abnormalities	1
Atrial Fibrillation	1
Subepicardial Injury	2

NOTE: Some subjects demonstrated more than one electrocardiographic abnormality, therefore total abnormalities will exceed the total number of subjects.

A-V block although this was the only Group III subject with this finding.

Two subjects demonstrated possible subepicardial injury; the first, a 22-year old white male had possible damage in the inferoseptal and anterolateral regions of the heart, and the second, a 16-year old white male was diagnosed as having possible diffuse subepicardial injury. This youth also demonstrated S-T segment changes.

Serial electrocardiograms were obtained on 15 of the 54 subjects in Group III at 24- and 48-hour intervals following the accident (Table 11).

Abnormal findings were reported in 13 of the 15 subjects receiving serial electrocardiograms.

An 18-year old white female demonstrated sinus tachycardia on the initial tracing which resided on subsequent tracings. Left ventricular hypertrophy was observed in this same subject on the second and third tracings.

Another 18-year old white female had a normal initial tracing, however a serial tracing taken 24 hours later showed evidence of phasic sinus arrhythmia.

A third subject, a 27-year old white male was found to have sinus tachycardia on the first tracing; however the second tracing was within normal limits.

A 30-year old white female and a 34-year old white female were found to have ectopic atrial pacemakers in the first electrocardiogram. This finding was retained in the second and third tracings on the younger woman; however, the older woman demonstrated a wandering pacemaker, sinus to high nodal, in subsequent tracings.

TABLE 11

GROUP III - ELECTROCARDIOGRAPHIC ABNORMALITIES
SERIAL TRACINGS

Abnormality	Number of Subjects with Abnormality
Ventricular Extrasystoles	1
Nonspecific T Wave Abnormalities	5
Ectopic Atrial Pacemaker	2
Wandering Pacemaker	1
Sinus Tachycardia	6
Subepicardial Injury	1
Sinus Bradycardia	2
Incomplete Bundle Branch Block (Right)	1
Complete Bundle Branch Block (Right)	2
Posteriodiaphragmatic myocardial infarction	1
Suggestive Posteriolateral Injury	1
Posterior Myocardial Ischemia	1
Left Ventricular Hypertrophy	1
Phasic Sinus Arrhythmia	1
Atrial Extrasystoles	1
Nodal Extrasystoles	1

NOTE: Some subjects demonstrated more than one electrocardiographic abnormality, therefore total abnormalities will exceed the total number of subjects.

A 20-year old white male, who was ambulatory in the hospital emergency room, was found to have an incomplete right bundle branch block in the first and second tracings, which developed into a complete right bundle branch block within 48 hours after the accident. This young man also demonstrated primary nonspecific T wave abnormalities.

An 11-year old white male was found to have a complete right bundle branch block with occasional extrasystoles in the initial tracing. The right bundle branch block receded on the serial tracings; however, primary nonspecific T wave abnormalities, nodal extrasystoles, and sinus tachycardia were all observed.

A 33-year old white male whose vehicle was hit by a train demonstrated some rather startling findings. The first tracing done soon after injury revealed not only sinus tachycardia, but a posteriodiaphragmatic myocardial infarction of uncertain duration with suggestive posteriolateral injury. The second tracing showed persistent but decreasing evidence of the posteriodiaphragmatic myocardial infarction, with any indication of posteriolateral injury now absent, and evidence of posterior ischemia present. The third electrocardiogram demonstrated evidence consistent with posteriodiaphragmatic myocardial infarction, and the indications of myocardial ischemia were now absent. This man was discharged from the hospital within a week, and it is worth noting that this individual had never been treated for, nor had ever experienced any cardiac symptoms.

Two subjects demonstrated persistent sinus tachycardia; one of these individuals, a 16-year old white male, had in addition, primary nonspecific T wave abnormalities, and evidence of anterolateral subepicardial injury, which disappeared in follow-up tracings.

A 15-year old white male and a 22-year old white Viet Nam veteran demonstrated sinus bradycardia. The younger subject had persistent bradycardia, as evidenced in serial electrocardiograms, and the older man retained the sinus bradycardia and developed primary nonspecific T wave abnormalities and both conditions persisted into the third tracing.

Ventricular extrasystoles were noted in only one subject, a 58-year old white male, who retained the condition in each of the three tracings obtained.

Primary nonspecific T wave abnormalities were noted in five subjects. In two subjects, these abnormalities of the T wave were noted in only the first tracing and in only one subject did a T wave abnormality, discovered on an initial tracing continue through all tracings. Primary nonspecific T wave abnormalities were noted, for the first time, in the second electrocardiograms of two subjects; however, in only one of the subjects just mentioned, did this condition persist into the third serial tracing.

Table 12 compares the findings on Group III subjects who received only one electrocardiogram with the findings on Group III subjects who received serial tracings.

Table 13 indicates the electrocardiographic abnormalities observed in Group III subjects and lists the number of subjects with single and serial tracings who demonstrated the particular electrocardiographic finding.

The median age level for Group I was 27 years. Positive findings or electrocardiographic abnormalities were observed with almost equal frequency above and below the median age level. Seven subjects

TABLE 12

COMPARISON OF ELECTROCARDIOGRAPHIC FINDINGS - BETWEEN SINGLE TRACINGS AND SERIAL TRACINGS GROUP III

Single ECG's Abnormal Findings	Single ECG's Normal Findings	Serial ECG's Abnormal Findings	Serial ECG's Normal Findings	Total ECG's Single and Serial
22	17	13	2	54

TABLE 13

COMPARISON OF ABNORMAL ELECTROCARDIOGRAPHIC FINDINGS BETWEEN SINGLE ECG'S AND SERIAL ECG'S IN GROUP III

Condition	Number of Subjects Demonstrating Condition in Single Tracings	Number of Subjects Demonstrating Condition in Single Tracings
Ventricular Extrasystoles	2	1
Nonspecific T Wave Abnormalities	7	5
Ectopic Atrial Pacemaker	0	2
Wandering Pacemaker	0	1
Sinus Tachycardia	5	6
Right Bundle Branch Block (Incomplete)	5	1
Left Ventricular Hypertrophy	4	1
Right Ventricular Hypertrophy	1	0
S-T Segment Abnormalities	1	0
Atrial Fibrillation	1	0
Subepicardial Injury	2	1
Complete RBBB	0	2
Posterior Diaphragmatic Myocardial Infarction	0	1
Suggestive Posteriolateral Injury	0	1
Posterior Myocardial Ischemia	0	1
Phasic Sinus Arrhythmia	0	1
Atrial Extrasystoles	0	1
Nodal Extrasystoles	0	1
TOTAL SUBJECTS*	22	13

*NOTE: Some subjects may have had more than one positive finding.

demonstrated electrocardiographic abnormalities in the age groups below the median age level and eight subjects were observed to have electrocardiographic abnormalities in the age groups above the median age level.

All subjects with electrocardiographic abnormalities below the median age level were under 21 years of age, while those subjects with electrocardiographic abnormalities above the median age level were distributed rather evenly throughout all subjects above the median age level.

Four males and three females below the median age level demonstrated electrocardiographic abnormalities. Four males and four females in the subgroup above the median age level demonstrated electrocardiographic abnormalities (Table 14).

A Chi-square analysis was done to determine if there was a significant difference in the number of subjects demonstrating electrocardiographic abnormalities below the median age level and the number of subjects with electrocardiographic abnormalities above the median age level. The Chi-square value of 0.07654 revealed no significant difference between the two subgroups, when tested at one degree of freedom.

The median age level for Group III was 25.5 years. Seventeen subjects were observed to have electrocardiographic abnormalities below the median age level as compared to 18 subjects who demonstrated electrocardiographic abnormalities above the median age level. The frequency of electrocardiographic abnormalities appears to be rather evenly distributed throughout all subjects in Group III (Table 15).

Fourteen males and three females below the median age level were found to have some electrocardiographic abnormality, and in the age group above the median age level, 13 males and 5 females were observed to

TABLE 14

GROUP I - ELECTROCARDIOGRAPHIC FINDINGS ON SUBJECTS BELOW AND ABOVE MEDIAN AGE LEVEL

Sex	Number of Subjects Below Median Age Level With Abnormal ECG Findings	Number of Subjects Below Median Age Level With Normal ECG Findings	Number of Subjects Above Median Age Level With Abnormal ECG Findings	Number of Subjects Above Median Age Level With Normal ECG Findings
Male	4	6	4	5
Female	3	2	4	2
Totals	7	8	8	7

TABLE 15

GROUP III - ELECTROCARDIOGRAPHIC FINDINGS ON SUBJECTS BELOW AND ABOVE MEDIAN AGE LEVEL

Sex	Number of Subjects Below Median Age Level With Abnormal ECG Findings	Number of Subjects Below Median Age Level With Normal ECG Findings	Number of Subjects Above Median Age Level With Abnormal ECG Findings	Number of Subjects Above Median Age Level With Normal ECG Findings
Male	14	9	13	4
Female	3	1	5	5
Totals	17	10	18	9

demonstrate electrocardiographic abnormalities.

A Chi-square analysis was done to determine if there was a significant difference in the number of subjects demonstrating electrocardiographic abnormalities below the median age level and the number of subjects with electrocardiographic abnormalities above the median age level. The Chi-square value of 0.0812 indicated no significant difference between the two subgroups, when tested at one degree of freedom.

The elapsed time in months between the motor vehicle accident and the electrocardiogram for Group I subjects ranged from 1 to 50 months.

The mean elapsed time was determined to be 25.4 months for all subjects in Group I, and 21.46 months for those Group I subjects found to have electrocardiographic abnormalities. The range, in months, for those persons demonstrating electrocardiographic abnormalities was between 2 and 48 months.

The median elapsed time in months for those subjects demonstrating electrocardiographic abnormalities was 23 months (Table 16).

The make and year of vehicles driven by Group I subjects was analyzed in an attempt to correlate the presence of electrocardiographic abnormalities with the year and make of vehicle. Only one Group I subject was driving a vehicle equipped with a collapsible steering column, and, therefore, it was impossible to attempt any correlation between the presence or absence of the collapsible steering column and the electrocardiographic findings.

Nine of 20 Group I subjects driving General Motors products demonstrated electrocardiographic abnormalities and the remaining 11 subjects had electrocardiograms which were within normal limits.

TABLE 16

GROUP I - ELAPSED TIME BETWEEN ACCIDENT AND ELECTROCARDIOGRAM

Subject	Age	Sex	Race	Time in Months	Electrocardiographic Finding
01 - L.B.	61	M	W	20	Normal
02 - J.G.	20	F	W	16	Normal
03 - C.Y.	23	M	W	28	Normal
04 - M.W.	21	M	W	23	Abnormal
05 - W.P.	37	M	N	30	Abnormal
06 - R.C.	67	M	W	25	Abnormal
07 - H.S.	51	M	W	23	Abnormal
08 - L.F.	38	F	W	31	Abnormal
09 - C.W.	21	M	N	17	Abnormal
10 - M.C.	64	F	W	22	Abnormal
11 - A.M.	47	F	W	9	Abnormal
12 - J.J.	35	F	W	24	Normal
13 - D.G.	20	F	W	17	Abnormal
14 - D.D.	24	M	W	36	Normal
15 - W.H.	40	M	W	26	Normal
16 - R.T.	42	F	W	14	Abnormal
17 - B.L.	36	F	W	50	Normal
18 - K.J.	16	F	N	10	Abnormal
19 - F.M.	24	M	W	1	Normal
20 - E.F.	38	M	W	24	Normal
21 - N.H.	19	F	W	28	Abnormal
22 - G.W.	20	M	W	24	Normal
23 - P.F.	24	M	W	49	Normal
24 - C.B.	20	F	W	2	Abnormal
25 - J.J.	42	M	W	38	Normal
26 - N.B.	50	M	W	45	Normal
27 - S.J.	25	M	W	21	Normal
28 - C.C.	20	M	W	23	Abnormal
29 - C.D.	28	F	W	38	Normal
30 - B.A.	29	M	W	48	Abnormal

The one subject driving a General Motors vehicle which was equipped with a collapsible steering column did show evidence of suggestive subepicardial injury on the inferoseptal and posterolateral heart surfaces almost 23 months after the accident.

Three of six subjects driving Ford Motor Company products were found to have electrocardiographic abnormalities with the remaining three subjects demonstrating normal electrocardiograms. None of the Ford vehicles in this group was equipped with the collapsible steering column.

Two Group I subjects were driving Chrysler products, neither of which was equipped with the collapsible steering column, and both drivers demonstrated electrocardiographic abnormalities.

The remaining Group I subject with an electrocardiographic abnormality was the driver of a 1966 Rambler, which was not equipped with the collapsible steering column, who reported severe chest pain following the accident and was found to have minor nonspecific T wave abnormalities (Table 17).

The make and year of vehicles driven by Group III subjects was analyzed in an attempt to correlate the presence of electrocardiographic abnormalities with the energy absorbing steering columns which were installed in 1967 General Motors, American Motors and Chrysler Corporation products and 1968 Ford Motor Company vehicles.

Seventeen of 24 subjects driving General Motors vehicles were discovered to have electrocardiographic abnormalities whereas only 7 subjects demonstrated normal electrocardiograms. Those with electrocardiographic abnormalities included 5 subjects whose vehicle was equipped with the collapsible steering column and 12 subjects whose vehicles were not

TABLE 17

ANALYSIS OF VEHICLES DRIVEN BY GROUP I SUBJECTS FOUND TO HAVE ELECTROCARDIOGRAPHIC ABNORMALITIES

Manufacturer	Make	Year	Equipped with Collapsible Steering Column	
			Yes	No
General Motors	Chevrolet	1956		X
General Motors	Chevrolet	1961		X
General Motors	Chevrolet	1962		X
General Motors	Chevrolet	1965		X
General Motors	Chevrolet	1965		X
General Motors	Chevrolet	1968	X	
General Motors	Oldsmobile	1960		X
General Motors	Oldsmobile	1962		X
General Motors	Pontiac	1965		X
Ford Motor Company	Comet	1961		X
Ford Motor Company	Ford	1963		X
Ford Motor Company	Mustang	1967		X
Chrysler Corp.	Plymouth	1964		X
Chrysler Corp.	Plymouth	1964		X
American Motors	Rambler	1966		X

so equipped.

Seven subjects, as mentioned, demonstrated no electrocardiographic abnormalities. Two of these subjects with normal tracings were driving G-M vehicles which were equipped with the collapsible column and five subjects with normal tracings were driving G-M vehicles which were not equipped with the collapsible steering column.

Nine of 16 subjects driving Ford products were found to have electrocardiographic abnormalities with the remaining 7 subjects demonstrating electrocardiograms which were within normal limits. None of the Ford drivers were operating vehicles equipped with the collapsible steering column. Two vehicles, a 1968 Ford Pickup truck and a 1969 Ford Pickup truck are included in this series of Ford vehicles; however, Ford Motor Company does not install collapsible steering columns in their pickup truck line.

Both pickup truck drivers demonstrated electrocardiographic abnormalities, and one of these drivers, a 20-year old white male demonstrated an incomplete right bundle branch block and minor nonspecific T wave abnormalities, which developed into a complete right bundle branch block by the third day after the accident.

Four of 7 subjects, in Group III, driving Chrysler products were discovered to have electrocardiographic abnormalities. Two of the four subjects with electrocardiographic abnormalities were driving vehicles which were equipped with the collapsible steering column and two were driving vehicles which were not so equipped.

The remaining subjects who were operating Chrysler products and were found to have no electrocardiographic abnormalities included two who

were driving vehicles equipped with the collapsible steering column and one subject whose vehicle was not so equipped.

Five subjects were operating Volkswagen vehicles. Three subjects demonstrated electrocardiographic abnormalities and in each case, the vehicle was not equipped with the collapsible steering column. The Volkswagen vehicles driven by the two remaining subjects who were found to have normal electrocardiograms were not equipped with a collapsible steering column.

One subject driving a 1962 Rambler which was not equipped with the collapsible steering column was found to have an incomplete right bundle branch block, and the remaining subject, a 16-year old white female driving a 1966 Renault Dauphine was discovered to have suggested left ventricular hypertrophy; the Renault was not equipped with the collapsible steering column (Table 18).

Tables 19 and 20 give a breakdown of all vehicles driven by Group I and III subjects according to the manufacturer and the reported electrocardiographic findings.

In summary, of the 54 subjects who comprise Group III, 35 were found to have some degree of electrocardiographic abnormality.

Seven subjects driving vehicles with collapsible steering columns were found to have electrocardiographic abnormalities.

Twenty-eight subjects demonstrated electrocardiographic abnormalities and were driving vehicles which were not equipped with the collapsible steering column.

Three subjects with normal electrocardiograms were driving vehicles equipped with the collapsible steering column, and 16 subjects

TABLE 18

ANALYSIS OF VEHICLES DRIVEN BY GROUP III SUBJECTS FOUND TO HAVE ELECTROCARDIOGRAPHIC ABNORMALITIES

Manufacturer	Make	Year	Equipped with Collapsible Steering Column	
			Yes	No
General Motors	Chevrolet	1959		X
General Motors	Corvair	1962		X
General Motors	Corvair	1962		X
General Motors	Chevrolet	1962		X
General Motors	Chevrolet	1963		X
General Motors	Chevrolet	1964		X
General Motors	Chevrolet	1964		X
General Motors	Corvair	1965		X
General Motors	Chevrolet	1966		X
General Motors	Corvair	1966		X
General Motors	Chevrolet	1967	X	
General Motors	Chevrolet	1968	X	
General Motors	Chevrolet	1969	X	
General Motors	Chevrolet	1969	X	
General Motors	Cadillac	1962		X
General Motors	Cadillac	1963		X
General Motors	Pontiac	1968	X	
Ford Motor Company	Falcon	1962		X
Ford Motor Company	Thunderbird	1963		X
Ford Motor Company	Mercury	1963		X
Ford Motor Company	Mercury	1963		X
Ford Motor Company	Galaxy	1964		X
Ford Motor Company	Cortina	1966		X
Ford Motor Company	Galaxy	1966		X
Ford Motor Company	Ford P/U	1968		X
Ford Motor Company	Ford P/U	1969		X
Chrysler Corporation	Dodge Van	1965		X
Chrysler Corporation	Dodge	1966		X
Chrysler Corporation	Plymouth	1968	X	
Chrysler Corporation	Chrysler	1968	X	
Volkswagen	Sedan	1960		X
Volkswagen	P/U	1962		X
Volkswagen	Sedan	1970		X
American Motor	Rambler	1962		X
Other Foreign	Renault	1966		X

TABLE 19

ANALYSIS OF ELECTROCARDIOGRAPHIC FINDINGS WITH REFERENCE TO THE AUTOMOBILE MANUFACTURER - GROUP I

Manufacturer	Number of Vehicles	Number of ECGS With Abnormalities	Number of ECGS Within Normal Limits
General Motors	20	9	11
Ford Motor Company	6	3	3
Chrysler Corp.	2	2	0
American Motors	1	1	0
Foreign	1	0	1
Totals	30	15	15

TABLE 20

ANALYSIS OF ELECTROCARDIOGRAPHIC FINDINGS WITH REFERENCE TO THE AUTOMOBILE MANUFACTURER - GROUP III

Manufacturer	Number of Vehicles	Number of ECGS With Abnormalities	Number of ECGS Within Normal Limits
General Motors	24	17	7
Ford Motor	16	9	7
Chrysler Corp.	7	4	3
American Motors	1	1	0
Volkswagen	5	3	2
Other Foreign	1	1	0
Totals	54	35	19

with normal electrocardiograms were driving vehicles which were not equipped with the collapsible steering column.

A Chi-square analysis based on the above information indicated that, for this sample, there was no significant difference in the frequency of electrocardiographic abnormalities found in subjects who were driving vehicles equipped with collapsible steering columns and those who were driving vehicles which were not equipped with the collapsible steering column (Table 21).

The absence of a statistically significant difference between the frequency of electrocardiographic abnormalities found in subjects who were drivers of vehicles equipped with collapsible steering columns and those who were driving vehicles which were not equipped with the device should not lead to the questionable conclusion that there is little difference between the efficacy of the collapsible steering column and its non-collapsible predecessor in preventing damage to the heart muscle.

Subjects were selected because they had been drivers in motor vehicle collisions, and not because it was known with certainty that they had actually struck the steering wheel assembly at impact, thus availing themselves of the collapsible design of the mechanism.

It must be further pointed out that the type and nature of the collision was not determined (head-on, rear end, side impact, etc.), thus supporting the position that no judgement can be drawn regarding the relative effectiveness or ineffectiveness of the collapsible steering column.

The crux of the inquiry lies in the realization that electrocardiographic abnormalities were discovered in many drivers who were

TABLE 21

A COMPARISON OF THE NUMBER OF SUBJECTS WITH AND WITHOUT ELECTROCARDIOGRAPHIC ABNORMALITIES AND THE PRESENCE OR ABSENCE OF THE ENERGY ABSORBING-COLLAPSIBLE STEERING WHEEL - GROUP III

	Collapsible Steering Column	Non-collapsible Steering Column
Subjects with Electro-cardiographic Abnormality	7	28
Subjects with Normal Electrocardiogram	3	16
Totals	10	44

Chi-square = 0.00265 @ 1 degree freedom: Not significant

believed to be free from any thoracic involvement resulting from trauma.

Data from Group I subjects were further analyzed in an attempt to determine the nature and extent of injuries received by those subjects who demonstrated electrocardiographic abnormalities and those subjects who were found to have normal electrocardiograms.

Emergency room reports or hospital records and discharge summaries were evaluated and the injuries rated on the Av-CIR-Injury Scale (Appendix A).

Fifteen of 30 Group I subjects demonstrated electrocardiographic abnormalities and 15 subjects had electrocardiograms which were within normal limits. Those with abnormalities included two subjects who were uninjured, six subjects who received only minor injuries and seven subjects who sustained severe injuries.

Those with normal electrocardiograms included five subjects with minor injuries, four subjects with moderate injuries, two subjects with severe injuries, two subjects with serious injuries and two subjects who sustained critical injuries.

Thirty-five of 54 Group III subjects were found to have electrocardiographic abnormalities. Eleven subjects sustained only trivial injuries or were not injured at all, ten subjects received only minor injuries, seven subjects were moderately injured, four subjects sustained severe injuries and three subjects received serious injuries. The remaining 19 subjects with normal electrocardiograms included eight subjects who received trivial injuries or were uninjured, ten subjects who sustained minor injuries, and one subject who was moderately injured.

Thirty-six subjects from Groups I and III, with Av-CIR ratings

of three or less, demonstrated electrocardiographic abnormalities. This finding is interesting in view of the fact that only 14 subjects from the same groups with abnormal electrocardiograms had Av-CIR ratings of "4" or more.

In addition, only three subjects, all of whom were from Group III, with Av-CIR ratings of five (serious but not dangerous injuries) were found to have electrocardiographic abnormalities.

No subjects sustaining critical injuries were found to have electrocardiographic abnormalities (Table 22).

Data were analyzed regarding the possible relationship between abnormal electrocardiographic findings and fractures of the ribs and/or sternum. Only seven subjects from Groups I and III demonstrated electrocardiographic abnormalities and fractures of the ribs and/or sternum. Forty-three subjects in Groups I and III demonstrated electrocardiographic abnormalities in the absence of sternal and/or rib fractures. One subject from Group I sustained multiple rib fractures but had a normal electrocardiogram, and the remaining 33 subjects from Groups I and III with normal electrocardiograms were all free from sternal and/or rib fractures (Table 23).

TABLE 22

BREAKDOWN OF Av-CIR RATINGS AND ELECTROCARDIOGRAPHIC FINDINGS-GROUP I AND GROUP III

	Av-CIR RATING SCALE						
GROUP I	1	2	3	4	5	6	Totals
Electrocardiograms - Normal Findings	0	5	4	2	2	2	15
Electrocardiograms - Abnormalities	2	6	0	7	0	0	15
							30
GROUP III							
Electrocardiograms - Normal Findings	8	10	1	0	0	0	19
Electrocardiograms - Abnormalities	11	10	7	4	3	0	35
							54

Av-CIR Rating Scale Legend:
1 - No injury or trivial injury
2 - Minor injury
3 - Moderate injury - not dangerous
4 - Severe injury - but not dangerous
5 - Serious - dangerous
6 - Critical - dangerous

TABLE 23

ELECTROCARDIOGRAPHIC FINDINGS AND THEIR RELATION TO STERNAL AND/OR RIB FRACTURES - GROUPS I AND III

Group	N	Subjects with Abnormal ECGS and Sternal and/or Rib Fracture	Subjects with Abnormal ECGS and no Sternal and/or Rib Fracture	Subjects with Normal ECG Findings and Sternal and/or Rib Fracture	Subjects with Normal ECG Findings and no Sternal and/or Rib Fracture
GROUP I	30	2	13	1	14
GROUP III	54	5	30	0	19
TOTALS	84	7	43	1	33

CHAPTER V

SUMMARY AND CONCLUSION

There is documented evidence to support the contention that the cardiovascular system may be injured by nonpenetrating mechanical trauma.

Lasky (76) quoting Ballance points out that in the eighteenth century it was first recognized that an individual might suffer a heart "wound" and continue to live without evidence of serious injury.

The literature indicates that while direct penetrating injuries are easily recognized, nonpenetrating injuries remained obscure and, for the most part, overlooked.

Lasky (76) goes on to point out that the position of the heart being in the middle mediastinum renders it readily susceptible to injury resulting from acceleration, deceleration or crushing.

It would be difficult, if not impossible, to determine the incidence of nonpenetrating heart trauma, because, as Lasky (76) indicates, "the mortality rate is low and the morbidity generally minimal". In addition, other physical injuries often mask the manifestation of cardiac trauma which should be searched for immediately after injury if it is to be discovered.

Lasky (76) reported that Parmley et al. found that the incidence of cardiac injury at autopsy and in a clinical series varied from 10 to 75 per cent, and Lasky further stated that when the lower percentage is

used as an index, the annual number of cardiac injuries from automobile accidents alone in the United States is in the range of 150,000 cases.

Moritz and Atkins (69), Bright and Beck (7) and Kissane, et al. (34) demonstrated with animals, that direct nonpenetrating cardiac trauma seldom produces serious consequences unless the myocardium ruptures or fatal arrhythmia developes.

Nonpenetrating thoracic trauma may produce cardiac tamponade, coronary artery occlusion, auricular flutter, ventricular fibrillation, traumatic pericarditis, purulent pericarditis, hemopericardium, myocardial rupture, aortic aneurysms, valvular lesions, and contusions of the heart. In addition, Lasky (76) states that a "vagosympathetic effect", or direct interference with the neuromuscular mechanism within the heart, presumably produces cardiac irritability (commotio cordis). This may result in ventricular arrhythmia or heart stoppage.

Less dramatic, but important, abnormalities which are believed to result from nonpenetrating thoracic trauma include the conditions which were observed in many of the subjects in this study, and they include possible left ventricular hypertrophy, A-V block, atrial enlargement, disorders of rhythm, subepicardial injury, disorders of rate, atrial fibrillation, nonspecific T wave abnormalities, bundle branch block, S-T segment abnormalities, and ventricular extrasystoles. Of course, it would be impossible to prove a definite causal relationship between the suspected trauma and the pathological condition; however, the repeated instances wherein the individual diagnoses were made time and time again, coupled with the fact that more accident victims demonstrated the specific abnormality than a matched control subject, supports the contention

that trauma is the most likely agent responsible for the electrocardiographic abnormalities.

Perhaps, one of the most interesting findings of this study was the relatively high incidence of incomplete and complete right bundle branch block. Johnson et al. (77), in an analysis of 67,375 apparently healthy adults, found 106 cases of complete right bundle branch block. This survey provided a unique opportunity to study this condition in an assumed disease-free population.

In Group III of this study, eight subjects demonstrated either an incomplete or a complete right bundle branch block. The incidence of right bundle branch block in Johnson's study was determined to be 1.5 cases per thousand; however, the incidence of right bundle branch block observed in this study, was determined to be 70.1 cases per thousand for the entire population (114 subjects) and 148 cases per thousand for Group III alone.

This finding gives credence to the possibility that the neuroconductive pathways of the heart muscle may be directly affected by nonpenetrating thoracic trauma, and in many motor vehicle accidents the driver may very well sustain an injury of this nature. It must be noted, however, that unless other evidence of heart disease is present, or unless the individual is in an older age group with a previously normal electrocardiogram, it should not be accepted as strict diagnostic evidence of significant underlying heart disease.

It has been the intention of this author to provide information which may be beneficial to clinicians as well as other workers in appraising accident victims in terms of hidden or masked cardiovascular damage

resulting from nonpenetrating blunt thoracic trauma which may often manifest itself in the absence of contusions, fractures or other visible evidence.

Lasky (76) has enumerated the most important symptoms, signs and laboratory findings that occur in cardiac injuries, as they are as follows:

SYMPTOMS: Precordial pain, dyspnea, orthopnea, cyanosis; evidence of shock (gray pallor, weakness, sweating and restlessness).

SIGNS: Cardiac arrhythmias; fluctuations in blood pressure, increase in the area of cardiac dullness (suggesting pericardial effusion or cardiac dilatation); paradoxical pulse; abnormal heart sounds, pericardial friction rub; cardiac murmurs if they appear suddenly or change abruptly; signs of cardiac failure, such as pulmonary edema or right heart failure.

LABORATORY FINDINGS: Electrocardiographic abnormalities such as auricular flutter, extrasystoles and other disorders of rhythm; abnormal P waves; change in the QRS complex; abnormalities in the S-T segment and T wave abnormalities.

Serum glutamic-oxalacetic transaminase (SGO-T) abnormality.

Roentgenographic abnormality, such as diffuse enlargement of the cardiac silhouette (as with hemopericardium). It must be pointed out that not infrequently, the chest x-ray may be normal.

In severe trauma, the sedimentation rate is increased and a polymorphonuclear leukocytosis is present.

Electrocardiograms were evaluated on the basis of the readers diagnosis. An electrocardiogram demonstrating any deviation from accepted

normal standards was considered abnormal and reported. An electrocardiogram which did not demonstrate any deviations from accepted normal standards was reported as within normal limits or normal.

While disagreement may arise regarding the validity of designating disorders of rate, such as sinus tachycardia and sinus bradycardia as true "abnormalities" it is the considered opinion of this author that these conditions represent some alteration in cardiac function, and although they may be of an inconsequential nature, they, nonetheless, were discovered to exist, and must be considered in the ultimate analysis of data.

On the basis of data accumulated in this study the following conclusions regarding heart damage resulting from nonpenetrating steering wheel impact are offered:

1. Cardiac damage resulting from bodily trauma occurs in a variable incidence. No relationship seems to exist between cardiac damage and the type or extent of injury. Age does not appear to be a factor in determining whether a subject will sustain damage to the heart and fractures of the ribs and/or sternum need not exist before abnormalities in cardiac function can be observed.
2. There was a statistically significant difference between the number of subjects with electrocardiographic abnormalities in Group I and the matched sample, Group II, which lends support to the hypothesis that heart damage may result from blunt thoracic trauma.
3. The number of Group III subjects with electrocardiographic abnormalities was almost twice as great as the number of subjects

with normal electrocardiograms, thereby supporting the contention that post-traumatic electrocardiographic abnormalities will be discovered if electrocardiograms are taken soon after an accident and repeated at intervals thereafter.

4. Changes in the heart, as evidenced by the electrocardiogram, may not occur immediately; therefore, recognition of early signs of cardiac injury is important so that proper clinical safeguards may be instituted to minimize complications and to ensure prompt and efficient treatment should more serious sequellae develop.
5. There is little doubt that many cases of damage to the heart resulting from nonpenetrating chest trauma are missed and not diagnosed, and therefore, the clinician must avoid overlooking cardiac injuries while treating those results of trauma more readily seen and diagnosed. It follows that clinical awareness will statistically increase the incidence of traumatic nonpenetrating heart disease.
6. The nature of the trauma itself sustained by subjects from Group I and Group III was of such a varying type and degree that a much larger series of subjects would be required before any statements could be made regarding any specific predilection to particular accidents which might cause heart injury. In addition, the sample size was too small and too little was known about the nature of the individual accident to make any definite statements regarding the relative efficacy of the collapsible steering column in attributing to or preventing heart damage.
7. Abnormalities such as sinus arrhythmias, sinus bradycardia and

sinus tachycardia were the most commonly diagnosed findings in this series. Nonpenetrating thoracic trauma seldom produces serious consequences unless the myocardium is ruptured or fatal arrhythmias develop. Cardiac tamponade, coronary artery occlusion, auricular flutter, ventricular fibrillation, traumatic pericarditis, hemopericardium, myocardial rupture, aneurysmal dilatation and valvular lesions were not observed in this series, however, the literature indicates that these sequels to blunt thoracic trauma have been reported.

8. The electrocardiogram is a guide in both diagnosis and treatment; however, the comparatively high proportion of abnormal tracings should not be accepted as absolute evidence that heart disease, of clinical significance, is common after trauma.

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APPENDIXES

APPENDIX A

DEGREES OF INJURY[a] USED BY Av-CIR

(Revised 8/58)

Degrees[b]

1 Trivial or None

2 Minor
"Minor" contusions, lacerations, abrasions in any area(s) of the body. Sprains, fractures, dislocations of fingers, toes, or nose. Dazed or slightly stunned. Mild concussion evidenced by mild headache, with no loss of consciousness.

3 Moderate - but not dangerous.
"Moderate" contusions, lacerations, abrasions in any area(s) of the body. Sprains of the shoulders or principal articulations of the extremities. Uncomplicated, simple or green-stick fractures of extremities and jaw. Concussion as evidenced by loss of consciousness not exceeding 5 minutes, without evidence of other intracranial injury.

4 Severe - but not dangerous. Survival normally assured.
Extensive lacerations without dangerous hemorrhage. Compound or comminuted fractures, or simple fractures with displacements. Dislocations of the arms, legs, shoulders or pelvisacral processes. Fractures of the facial bones. Severe sprains of the cervical spine. Fracture of transverse and/or spinous processes of the spine, without evidence of spinal cord damage. Simple fractures of vertebral bodies of the dorsal and/or lumbar spine, without evidence of spinal cord damage. Compression fractures of L-3-4-5. Skull fracture without evidence of concussion or other intracranial injury. Concussion as evidenced by loss of consciousness of over 5 and up to 30 minutes, without evidence of other intracranial injury.

5 Serious - dangerous, but survival probable.
Lacerations with dangerous hemorrhage. Simple fractures of vertebral bodies of the cervical spine, without evidence of spinal cord damage. Compression fractures of vertebral bodies of dorsal spine and/or of L-1 and L-2, without evidence of spinal cord damage. Crushing or multiple fractures of the extremities and/or

of the chest. Indication of moderate intra-thoracic or intra-abdominal injury. Skull fracture with concussion as evidenced by loss of consciousness up to 30 minutes. Concussion as evidenced by loss of consciousness of over 30 minutes to 2 hours, without evidence of other intracranial injury.

6 Critical - dangerous, survival uncertain or doubtful. (Includes fatal terminations beyond 24 hours.)
Evidence of dangerous intrathoracic or intra-abdominal injury. Fractures or dislocations of vertebral bodies of cervical spine with evidence of cord damage. Compression fractures of vertebral bodies of dorsal spine, and/or L-1, L-2, with evidence of spinal cord damage. Skull fracture with concussion as evidenced by loss of consciousness beyond 30 minutes. Concussion as evidenced by loss of consciousness beyond 2 hours. Evidence of critical intracranial injury.

7 Fatal - within 24 hours of accident.
Fatal lesions in single region of the body, with or without other injuries to the 4th degree.

8 Fatal - within 24 hours of accident.
Fatal lesions in single region of the body, with other injuries to 5th or 6th degree.

9 Fatal
Fatal lesions in two regions of the body, with or without other injuries elsewhere.

10 Fatal
Fatal lesions in three or more regions - up to and including demolition of the body.

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[a]Based on observations during first 48 hours after injury and previously normal life expectancy.

[b]Degrees of total injury.

APPENDIX B

GROUP I - ELECTROCARDIOGRAMS - REPORTS AND DIAGNOSIS

Case # 01 - L.B. 61/w/m - 1965 Oldsmobile - Av-CIR = 5
Atrial Rate: 78
Ventricular Rate: 78
PR: .20
QRS: .06
QT: .32
T III is inverted; within normal limits

Case # 02 - J.S.G. - 20/w/f - 1966 Corvair - Av-CIR = 6
Atrial Rate: 75
Ventricular Rate: 75
PR: .13
QRS: .08
QT: .36
Within normal limits

Case # 03 - C.Y. - 23/w/m - 1962 Chevrolet - Av-CIR = 3
Atrial Rate: 82
Ventricular Rate: 82
PR: .16
QRS: .08
QT: .34
Within normal limits

Case # 04 - M.W. - 21/w/m - 1965 Chevrolet - Av-CIR = 4
Atrial Rate: 70
Ventricular Rate: 70
PR: .16
QRS: .06
QT: .36
Sinus arrhythmia
Right axis deviation

Case # 05 - W.P. - 37/n/m - 1965 Chevrolet - Av-CIR = 2
Atrial Rate: 68
Ventricular Rate: 68
PR: .24
QRS: .10
QT: .34
First degree A-V block/suggestive Left ventricular hypertrophy

Case # 06 - R.C. - 67/w/m - 1962 Chevrolet - Av-CIR = 4
Atrial Rate: 63
Ventricular Rate: 63
PR: .18
QRS: .08
QT: .40
Suggestive Left Ventricular hypertrophy

Case # 07 - H.S. - 51/w/m - 1966 Rambler - Av-CIR = 2
Atrial Rate: 72
Ventricular Rate: 72
PR: .16
QRS: .08
QT: .36
Left axis deviation; transition is abrupt at V III. T wave amplitude is low. 1 - LAD 2. Minor, nonspecific T wave abnormalities.

Case # 08 - L.F. - 38/w/f - 1961 Chevrolet - Av-CIR = 4
Atrial Rate: 83
Ventricular Rate: 83
PR: .14
QRS: .08
QT: .36
The p wave suggests atrial enlargement but are not necessarily diagnostic of same.

Case # 09 - C.W. - 21/n/m - 1961 Comet - Av-CIR =2
Atrial Rate: 75
Ventricular Rate: 75
PR: .16
QRS: .06
QT: .32
Sinus arrhythmia

Case # 10 - M.C. - 1962 Oldsmobile - Av-CIR = 4
Atrial Rate: 76
Ventricular Rate: 76
PR: .18
QRS: .06
QT: .34
Sinus arrhythmia

Case # 11 - A.M. - 47/w/f - 1965 Pontiac - Av-CIR = 2
Atrial Rate: 76
Ventricular Rate: 76
PR: .10
QRS: .06
QT: .34
Lown-Ginong-Levin Syndrome if patient has had bouts of tachycardia, otherwise coronary nodal rhythm.

Case # 12 - J.J. - 35/w/f - 1964 Renault - Av-CIR = 2
Atrial Rate: 72
Ventricular Rate: 72
PR: .16
QRS: .04
QT: .32
Low voltage - within normal limits

Case # 13 - D.G. - 20/w/f - 1964 Plymouth - Av-CIR = 4
Atrial Rate: 83
Ventricular Rate: 83
PR: .18
QRS: .06
QT: .32
A-V nodal rhythm (coronary sinus rhythm)

Case # 14 - D.D. - 20/w/m - 1954 Chevrolet Corvette - Av-CIR = 6
Atrial Rate: 77
Ventricular Rate: 77
PR: .14
QRS: .08
QT: .36
Within normal limits

Case # 15 - W.H. - 40/w/m - 1962 Chevrolet Corvair - Av-CIR = 2
Atrial Rate: 64
Ventricular Rate: 64
PR: .16
QRS: .08
QT: .36
Within normal limits

Case # 16 - R.T. - 42/w/f - 1964 Plymouth - Av-CIR = 4
Atrial Rate: 55
Ventricular Rate: 55
PR: .16
QRS: .06
QT: .36
Left axis deviation. Sinus bradycardia. There is poor R wave progression across the precordium.

Case # 17 - B.L. - 36/w/f - 1964 Ford - Av-CIR = 4
Atrial Rate: 75
Ventricular Rate: 75
PR: .16
QRS: .08
QT: .34
Within normal limits

Case # 18 - K.J. - 16/n/f - 1960 Oldsmobile - Av-CIR = 1
Atrial Rate: 95

Ventricular Rate: 95
PR: .16
QRS: .08
QT: .36
There is minimal S-T segment elevation in leads II, III, aVF, V5 and V6. This may represent a normal varient. T waves are inverted to diphasic in V1, V2 and V3. This could also be a normal varient or a retention of the juvenile pattern.

Case # 19 - F.M. - 24/w/m - 1959 Chevrolet - Av-CIR = 2
Atrial Rate: 66
Ventricular Rate: 66
PR: .14
QRS: .06
QT: .32
Within normal limits

Case # 20 - E.F. - 38/w/m - 1954 Chevrolet P/U - Av-CIR = 2
Atrial Rate: 75
Ventricular Rate: 75
PR: .16
QRS: .10
QT: .34
Amplitude of V2 is less than V1 - this could be electrode placement.
Within normal limits

Case # 21 - N.H. - 19/w/f - 1964 Ford - Av-CIR = 2
Atrial Rate: 88
Ventricular Rate: 88
PR: .16
QRS: .06
QT: .32
Premature nodal contraction - V6

Case # 22 - G.W. - 20/w/m - 1959 Chevrolet - Av-CIR = 5
Atrial Rate: 95
Ventricular Rate: 95
PR: .21
QRS: .08
QT: .34
Slightly prolonged P-R interval although not a true A-V block.
Within normal limits

Case # 23 - P.F. - 24/w/m - 1963 Chevrolet - Av-CIR = 3
Atrial Rate: 92
Ventricular Rate: 92
PR: .14

QRS: .08
QT: .32
Within normal limits

Case # 24 - C.B. - 20/w/f - 1967 Ford Mustang - Av-CIR = 4
Atrial Rate: 80
Ventricular Rate: 80
PR: .17
QRS: .08
QT: .34
Small q waves in II, III and aVF. T wave changes in V1, V2, V3, V4, and V5. All compatible with pulmonary embolic phenomena.

Case # 25 - J.J. - 42/w/m - 1964 Ford - Av-CIR = 3
Atrial Rate: 60
Ventricular Rate: 60
PR: .14
QRS: .10
QT: .36
Within normal limits

Case # 26 - S.J. - 25/w/m - 1966 Ford Mustang - Av-CIR = 3
Atrial Rate: 80
Ventricular Rate: 80
PR: .16
QRS: .08
QT: .36
Within normal limits

Case # 27 - N.B. - 50/w/m - 1966 Pontiac - Av-CIR = 2
Atrial Rate: 78
Ventricular Rate: 78
PR: .16
QRS: .08
QT: .30
Within normal limits

Case # 28 - C.C. - 20/w/m - 1968 Chevrolet - Av-CIR = 2
Atrial Rate: 77
Ventricular Rate: 77
PR: .14
QRS: .10
QT: .34
There is minimal S-T segment elevation in leads II, III, aVF, V5 and V6. This may represent a normal varient and previous tracings would be valuable, otherwise subepicardial injury in the inferoseptal and posterolateral surfaces of the heart must be considered.

Case # 29 - C.D. - 24/w/f - 1960 Pontiac - Av-CIR = 4
Atrial Rate: 72
Ventricular Rate: 72
PR: .12
QRS: .06
QT: .36
Within normal limits

Case # 30 - B.A. - 29/w/m - 1956 Chevrolet - Av-CIR = 1
Atrial Rate: 104
Ventricular Rate: 104
PR: .16
QRS: .10
QT: .32
Sinus tachycardia

APPENDIX C

GROUP II - ELECTROCARDIOGRAMS - REPORTS AND DIAGNOSIS

Case # 001 - J.N. - 56/w/m
Atrial Rate: 58
Ventricular Rate: 58
PR: .12
QRS: .06
QT: .36
Sinus Bradycardia

Case # 002 - D.F. - 21/w/f
Atrial Rate: 94
Ventricular Rate: 94
PR: .16
QRS: .06
QT: .32
The S-T segments are elevated 1 mm in leads I, aVL, V1 and V2.
This could represent a normal varient or an epicardial injury shift

Case # 003 - T.L. - 22/w/m
Atrial Rate: 92
Ventricular Rate: 92
PR: .16
QRS: .08
QT: .34
Within normal limits

Case # 004 - D.P. - 21/w/m
Atrial Rate: 94
Ventricular Rate: 94
PR: .14
QRS: .08
QT: .32
Within normal limits

Case # 005 - D.H. - 38/n/m
Atrial Rate: 75
Ventricular Rate: 75
PR: .18
QRS: .06
QT: .36
Sinus arrhythmia

Case # 006 - F.K. - 65/w/m
Atrial Rate: 86
Ventricular Rate: 86
PR: .14
QRS: .10
QT: .34
Left axis deviation. There is a 0.5 to 0.75 mm S-T segment depression in leads II, aVF, V4, V5 and V6 which are compatible with subendocardial ischemia.

Case # 007 - J.G. - 51/w/m
Atrial Rate: 82
Ventricular Rate: 82
PR: .16
QRS: .06
QT: .30
Within normal limits

Case # 008 - J.E. - 35/w/f
Atrial Rate: 100
Ventricular Rate: 100
PR: .16
QRS: .06
QT: .32
There is a "u" wave in leads V1 - V4 / Sinus tachycardia

Case # 009 - B.W. - 22/n/m
Atrial Rate: 76
Ventricular Rate: 76
PR: .16
QRS: .08
QT: .28
Within normal limits

Case # 010 - M.E. - 65/w/f
Atrial Rate: 77
Ventricular Rate: 77
PR: .14
QRS: .06
QT: .36
Transition is abrupt at V3. An old anteroseptal myocardial infarction cannot be excluded, although this tracing is not diagnostic.

Case # 011 - M.A. - 47/w/f
Atrial Rate: 72
Ventricular Rate: 72
PR: .20
QRS: .08
QT: .36
Within normal limits

Case # 012 - J.B. - 30/w/f
Atrial Rate: 78
Ventricular Rate: 78
PR: .12
QRS: .06
QT: .32
Within normal limits

Case # 013 - M.B. - 25/w/f
Atrial Rate: 64
Ventricular Rate: 64
PR: .14
QRS: .08
QT: .36
Within normal limits

Case # 014 - G.C. - 26/w/m
Atrial Rate: 64
Ventricular Rate: 64
PR: .16
QRS: .08
QT: .36
Within normal limits

Case # 015 - J.B. - 42/w/m
Atrial Rate: 65
Ventricular Rate: 65
PR: .16
QRS: .08
QT: .36
Within normal limits

Case # 016 - J.H. - 40/w/f
Atrial Rate: 64
Ventricular Rate: 64
PR: .14
QRS: .08
QT: .36
Within normal limits

Case # 017 - L.W. - 36/w/f
Atrial Rate: 78
Ventricular Rate: 78
PR: .12
QRS: .08
QT: .36
Within normal limits

Case # 018 - R.P. - 16/n/f
Atrial Rate: 66
Ventricular Rate: 66

PR: .16
QRS: .08
QT: .36
Within normal limits

Case # 019 - K.B. - 29/w/m
Atrial Rate: 68
Ventricular Rate: 68
PR: .16
QRS: .08
QT: 36
Within normal limits

Case # 020 - B.R. - 43/w/m
Atrial Rate: 50
Ventricular Rate: 50
PR: .16
QRS: .06
QT: .38
Sinus bradycardia

Case # 021 - L.B. - 21/w/f
Atrial Rate: 86
Ventricular Rate: 86
PR: .16
QRS: .08
QT: .32
Within normal limits

Case # 022 - B.F. - 22/w/m
Atrial Rate: 80
Ventricular Rate: 80
PR: .12
QRS: .06
QT: .32
Within normal limits

Case # 023 - J.L. - 28/w/m
Atrial Rate: 77
Ventricular Rate: 77
PR: .20
QRS: .10
QT: .32
Within normal limits

Case # 024 - K.J. - 22/w/f
Atrial Rate: 72
Ventricular Rate: 72
PR: .16
QRS: .08
QT: .34
Sinus arrhythmia

Case # 025 - K.P. - 40/w/m
Atrial Rate: 88
Ventricular Rate: 88
PR: .16
QRS: .08
QT: .32
Within normal limits

Case # 026 - K.B. - 49/w/m
Atrial Rate: 63
Ventricular Rate: 63
PR: .16
QRS: .08
QT: .36
Within normal limits

Case # 027 - K.V. - 26/w/m
Atrial Rate: 70
Ventricular Rate: 70
PR: .16
QRS: .08
QT: .32
Within normal limits

Case # 028 - G.S. - 20/w/m
Atrial Rate: 62
Ventricular Rate: 62
PR: .16
QRS: .08
QT: .36
Within normal limits

Case # 029 - S.K. - 30/w/f
Atrial Rate: 90
Ventricular Rate: 90
PR: .16
QRS: .08
QT: .34
Within normal limits

Case # 030 - K.T. - 28/w/m
Atrial Rate: 80
Ventricular Rate: 80
PR: .18
QRS: .08
QT: .32

APPENDIX D

GROUP III - ELECTROCARDIOGRAMS - REPORTS AND DIAGNOSIS

Case # 1 - G.A. - 36/w/m - 1969 Buick - Av-CIR = 2
Rate is 74 per minute.
PR: .14
QRS: .10
QT: .36
Sinus rhythm / S wave is present in I and aVL and persists through V5
Normal electrocardiogram

Case # 2 - W.A. - 58/w/m - 1966 Dodge - Av-CIR = 4
Tracing 1 - Rate is 80 per minute
PR: .15
QRS: .08
QT: .37
Sinus rhythm
Frequent ventricular extrasystoles, unifocal

Tracing 2 - Rate is 75 per minute.
PR: .14
QRS: .08
QT: .40
Sinus rhythm / voltage of T has decreased slightly in II, III, aVF and V6 through vB with slight sagging of the ST segment.
Frequent ventricular extrasystoles
Nonspecific T wave abnormalities

Tracing 3 - Rate is 77 per minute
PR: .16
QRS: .08
QT: .35
Sinus rhythm / voltage of T has increased in II, III and aVF
Recurrent ventricular extrasystoles

Case # 3 - F.A. - 34/w/f - 1967 Chevrolet - Av-CIR = 2
Rate is 78 per minute
PR: .15
QRS: .09
QT: .34
Sinus rhythm
Technical interference incident to muscle tremor is present.

P waves vary in form, and ventricular rhythm is slightly irregular
Wandering pacemaker, sinus to high nodal.

Tracing 2 - Rate is 105 per minute
PR: .13
QRS: .08
QT: .32
Sinus rhythm
P wave is now negative in III and low to flat in aVF.
QRS is essentially unchanged. Ectopic Atrial Pacemaker.

Case # 4 - G.A. - 16/w/m - 1969 Chevrolet - Av-CIR = 5
Rate is 105 per minute
PR: .12
QRS: .09
QT: .33
Sinus rhythm
T is negative in V4R and V1, and notched in V2 through V4
Very slight positive ST shift is present in I, II, and V5 through VB
Sinus tachycardia
Nonspecific T wave abnormalities
Possible anterolateral subepicardial injury.

Tracing 2 - Rate is 112 per minute
PR: .12
QRS: .08
QT: .32
Sinus rhythm
Sinus tachycardia

Case # 5 - J.B. - 43/w/m - 1966 Ford - Cortina - Av-CIR = 4
Rate is 115 per minute
PR: .12
QRS: .10
QT: .30
T is low to negative in I, II, aVL, aVF and V3 through VB
Sinus tachycardia
Primary nonspecific T wave abnormalities

Case # 6 - R.B. - 22/w/m - 1964 Ford - Av-CIR = 2
Rate is 70 per minute
PR: .14
QRS: .08
QT: .36
Sinus rhythm
Positive peaking of T is present in V2 through V4
Primary nonspecific T wave abnormalities

Tracing 2 - Rate is 53 per minute
PR: .12
QRS: .08
QT: .42

Sinus rhythm
Rate is decreased, and QT interval has increased, but is within normal limits. Positive peaking of T remains present in V2 through V4.
Sinus bradycardia
Primary nonspecific T wave abnormalities

Tracing 3 - Rate is 47 per minute
PR: .12
QRS: .08
QT: .44
Sinus rhythm
Rate has slowed but complexes are otherwise unchanged
Nonspecific T wave abnormalities, unchanged
Sinus bradycardia

Case # 7 - M.B. - 16/w/f - 1966 Renault - Av-CIR = 1
Rate is 74 per minute
PR: .12
QRS: .08
QT: .36
Sinus rhythm
Voltage on S prominent over the right precordium, but R is prominent in voltage in V4 through VB.
Suggested left ventricular hypertrophy

Case # 8 - L.B. - 22/w/m - 1957 Chevrolet - Corvette - Av-CIR = 3
Rate is 82 per minute
PR: .16
QRS: .08
QT: .32
Sinus rhythm
Normal electrocardiogram

Case # 9 - R.B. - 20/w/m - 1968 Ford P/U - Av-CIR = 2
Rate is 90 per minute
PR: .14
QRS: .09
QT: .35
Sinus rhythm
A late S is present in I, II, III, aVL, aVF and V1 through V8, with an RR' in V1.
Incomplete right bundle branch block

Tracing 2 - Rate is 70 per minute
PR: .15
QRS: .10
QT: .36
Sinus rhythm
Positive peaking of T has increased slightly in V2 through V6.
RR' is now absent in V1, but the late S persists through V6.
Incomplete right bundle branch block.
Primary nonspecific T wave abnormalities.

Tracing 3 - Rate is 68 per minute
PR: .16
QRS: .09
QT: .36
Sinus rhythm
Complexes are essentially unchanged
Complete right bundle branch block
Nonspecific T wave abnormalities.

Case # 10 - H.B. - 37/w/m - 1962 Cadillac - Av-CIR = 1
Rate is 105 per minute
PR: .15
QRS: .08
QT: .32
Sinus rhythm
Sinus tachycardia

Case # 11 - D.C. - 19/w/m - 1967 Comet - Av-CIR = 1
Rate is 74 per minute
PR: .16
QRS: .10
QT: .36
Sinus rhythm
A broad S is present in I, II, aVL and V1 through V8
Incomplete right bundle branch block
Suggestive left ventricular hypertrophy

Case # 12 - C.C. - 20/w/m - 1964 Ford - Av-CIR = 1
Rate is 84 per minute
PR: .16
QRS: .10
QT: .34
The mean QRS vector is +120°, with a T wave vector of +30°
RR' is absent in V4R and V1, but R is prominent in V4R
In the precordial leads a late S persists through V8
Possible right ventricular hypertrophy.

Case # 13 - L.C. - 29/w/f - 1966 Ford - Av-CIR = 2
Rate is 94 per minute
PR: .12
QRS: .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Case # 14 - M.C. - 36/w/m - 1968 Plymouth - Av-CIR = 1
Rate is 60 per minute
PR: .12
QRS: .10
QT: .36
A late S is present in I, II, III, aVL, aVF, and V2 through V9 with RR' in V1. Incomplete right bundle branch block.

Case # 15 - I.C. - 70/w/f - 1959 Chevrolet - Av-CIR = 2
Rate is 74 per minute
PR: .16
QRS: .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Case # 16 - D.D. - 25/w/m - 1966 Dodge - Av-CIR = 2
Rate is 60 per minute
PR: .13
QRS: .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Case # 17 - J.A. - 34/w/m - 1959 Chevrolet - Av-CIR = 5
Rate is 122 per minute
PR: .16
QRS: .08
QT: .30
Sinus rhythm
Q-S deflections are present in II, III aVF, with a mean QRS vector of -85°; the mean T wave vector is +60°.
Voltage of R decreases in V4 and V5, with an appearance of significant Q waves in V6 through VB.
Slight positive S-T shift is present in V8 through VB.
Sinus tachycardia
Posteriodiaphragmatic myocardial infarction, duration uncertain, but with suggestive posteriolateral injury.

Tracing 2 - Rate is 90 per minute
PR: .15
QRS: .08
QT: .34
Sinus rhythm
Slight clockwise rotation in the mean QRS vector has occurred, and voltage of R has increased in V2 through VB.
ST is now isoelectric, and T is low to flat in V7 and V8, and late negative in V9 and VB.
Persistent but decreasing evidence of posteriodiaphragmatic myocardial infarction.
Note: evidence of posteriolateral injury is now absent, now with suggestive posterior ischemia.

Tracing 3 - Rate is 105 per minute
PR:
QRS:
QT:
Sinus rhythm
T waves are now positive in V7 through VB

Persistent evidence consistent with posteriodiaphragmatic myocardial infarction.
Note: evidence of myocardial ischemia is now absent.

Case # 18 - D.E. - 16/w/m - 1963 Cadillac - Av-CIR = 2
Rate is 62 per minute
PR: .13
QRS: .08
QT: .38
Sinus rhythm
Slight positive S-T shift is present in I, II, aVF and V4 through V8.
Minor S abnormalities requiring clinical correlation.
Diffuse subepicardial injury is to be considered.

Case # 19 - J.E. - 18/w/f - 1962 Ford - Falcon - Av-CIR =3
Rate is 102 per minute
PR: .12
QRS: .08
QT: .30
Sinus rhythm
Sinus tachycardia

Tracing 2 - Rate is 65 per minute
PR: .12
QRS: .08
QT: .36
Sinus rhythm
Voltage of S is normal over the right precordium, but voltage of R is prominent in V4 through VB, with an intrinsicoid deflection of .05 seconds in V4.
Normal electrocardiogram.
Left ventricular hypertrophy cannot be excluded.

Tracing 3 - Rate is 66 per minute
PR: .12
QRS: .08
QT: .34
Sinus rhythm
Voltage of S is normal over the right precordium.
Voltage of R remains prominent through VB
Left ventricular hypertrophy cannot be excluded

Case # 20 - C.E. - 15/w/m - 1963 Mercury - Av-CIR = 2
Rate is 50 per minute
PR: .12
QRS: .10
QT: .44
Sinus rhythm
Sinus bradycardia

Tracing 2 - Rate is 52 per minute
PR: .12
QRS: .09
QT: .44
Sinus rhythm
Complexes are essentially unchanged
Sinus Bradycardia

Case # 21 - P.E. - 18/w/m - 1960 Volkswagen - Av-CIR = 1
Rate is 78 per minute
PR: .14
QRS: .10
QT: .36
Sinus rhythm
A late S is present in I, II, aVL and aVF, and persists through V6.
Voltage of R is prominent in V3 through VB, with an intrinsicoid deflection of 0.05 seconds duration in V3.
RR' is seen in aVR and V4R.
Incomplete right bundle branch block.
Left ventricular hypertrophy.

Case # 22 - D.F. - 22/w/m - 1964 Chevrolet - Av-CIR=2
Rate is 70 per minute
PR: .18
QRS: .11
QT: .40
Sinus rhythm
Slight positive ST shift is present in II, III, aVF and V3 through V7, with positive peaking of T in V3 through V6.
Nonspecific T wave abnormalities
Subepicardial injury in the inferoseptal and anterolateral regions cannot be excluded.

Case # 23 - G.F. - 75/w/m - 1966 Chevrolet - Av-CIR = 2
Rate is 72 per minute
PR: .17
QRS: .08
QT: .36
Sinus rhythm
Occasional ventricular extrasystoles

Case # 24 - D.F. - 16/w/m - 1962 Chevrolet - Av-CIR =3
Rate is 82 per minute
PR: .22
QRS: .08
QT: .32
Sinus rhythm
PR interval is prolonged. Complexes are otherwise normal
First degree A-V block, with otherwise normal electrocardiogram.

Case # 25 - M.G. - 16/w/m - 1968 Chevrolet - Av-CIR = 2
Rate is 67 per minute
PR: .15
QRS: .08
QT: .34
Sinus rhythm
T is low in I and negative in aVL
Minor nonspecific T wave abnormalities.

Case # 26 - B.N. - 32/w/m - 1969 Chevrolet - Av-CIR = 2
Rate is 67 per minute
PR: .17
QRS? .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Case # 27 - R.M. - 29/w/m - 1963 Mercury - Av-CIR = 1
Rate is 105 per minute
PR: .16
QRS: .08
QT: .30
Sinus rhythm
Sinus tachycardia

Case # 28 - M.M. - 17/w/m - 1962 Chevrolet - Corvair - Av-CIR = 2
Rate is 75 per minute
PR: .19
QRS: .08
QT: .38
Sinus rhythm
U waves are present in V3 through V5.
Voltage of S is prominent over the right precordium, voltage of R remains prominent at VB.
Intrinsicoid deflection in V5 of 0.05 seconds.
Suggestive left ventricular hypertrophy.

Case # 29 - B.M. - 19/w/f - 1965 Volkswagen - Av-CIR = 1
Rate is 88 per minute
PR: .18
QRS: .09
QT: .34
Sinus rhythm
Normal electrocardiogram

Case # 30 - C.M. - 42/w/m - 1962 Rambler - Av-CIR = 3
Rate is 82 per minute
PR: .16
QRS: .10
QT: .32
Sinus rhythm
A late S is present in I, aVL and persists through V6.
Incomplete right bundle branch block.

Case # 31 - C.M. - 18/w/m - 1967 Plymouth - Barracuda - Av-CIR = 2
Rate is 75 per minute
PR: .14
QRS: .08
QT: .40
Sinus rhythm
Normal electrocardiogram

Case # 32 - K.K. - 56/w/m - 1968 Chrysler - Av-CIR = 2
Rate is 45 per minute
PR: .--
QRS: .08
QT: .42
P waves are absent and ventricular rhythm is irregular
Atrial fibrillation with an average ventricular rate of 45 per minute.
Nonspecific T wave abnormalities.

Case # 33 - J.J. - 34/w/m - 1969 Ford - P/U - Av-CIR = 3
Rate is 72 per minute
PR: .13
QRS: .10
QT: .36
Complexes are deformed by uncontrollable patient motion.
T waves are low and notched in V3 through V6.
Voltage of R is prominent in V3 through V6, but S is normal over the right precordium, and voltage of R is normal in the back.
Primary nonspecific T wave abnormalities.

Case # 34 - G.J. - 17/w/m - 1963 Chevrolet - Av-CIR = 2
Rate is 77 per minute
PR: .12
QRS: .08
QT: .34
Sinus rhythm
Normal electrocardiogram

Case # 35 - W.H. - 20/w/m - 1967 Volkswagen - Av-CIR = 1
Rate is 64 per minute
PR: .12
QRS: .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Tracing 2 - Rate is 70 per minute
PR: .14
QRS: .08
QT: .34
Sinus rhythm
Complexes are essentially unchanged
Normal electrocardiogram

Case # 36 - S.H. - 28/N/m - 1965 Dodge - Van - Av-CIR = 1
Rate is 78 per minute
PR: .15
QRS: .10
QT: .33
Sinus rhythm
A broad S is present in I, II, aVL, aVF, and V1 through V6.
RR' is present in V4.
Incomplete right bundle branch block.

Case # 37 - T.H. - 18/w/m - 1966 Mustang - Av-CIR = 2
Rate is 65 per minute
PR: .12
QRS: .08
QT: .40
Sinus rhythm
Normal electrocardiogram

Case # 38 - L.H. - 27/w/m - 1966 Chevrolet - Corvette - Av-CIR = 3
Rate is 105 per minute
PR: .13
QRS: .09
QT: .32
Sinus rhythm
The mean QRS vector is approximately +100°. Ventricular rhythm is regular.
Complexes are deformed slightly by superimposed patient motion.
Sinus tachycardia.

Tracing 2 - Rate is 78 per minute
PR: .13
QRS: .10
QT: .33
Sinus rhythm
Slight counterwise rotation in the mean QRS vector has occurred.
Complexes are otherwise unchanged.

Case # 39 - E.P. - 34/w/f - 1964 Ford - Av-CIR = 2
Rate is 84 per minute
PR: .16
QRS: .08
QT: .32
Sinus rhythm
Normal electrocardiogram

Case # 40 - D.P. - 18/w/f - 1970 Volkswagen - Av-CIR = 1
Rate is 68 per minute
PR: .13
QRS: .09
QT: .37
Sinus rhythm
A late S is present in I, II, aVF, aVL and V1 through V6.
T is notched in V2
Normal electrocardiogram.

Tracing 2 - Rate is 88 per minute
PR: .13
QRS: .08
QT: .40
Sinus rhythm
Phasic sinus arrhythmia with otherwise normal electrocardiogram.

Case # 41 - J.R. - 26/w/m - 1958 Ford - Av-CIR = 1
Rate is 90 per minute
PR: .15
QRS: .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Case # 42 - B.R. - 51/w/m - 1962 Volkswagen - P/U - Av-CIR = 1
Rate is 72 per minute
PR: .16
QRS: .09
QT: .38
Sinus rhythm
A broad Q is present in II, and V7 through VB. The first point, 0.04 seconds duration of the QRS in III and aVF is slurred.
Voltage of T is low in I and aVL.
Nonspecific T wave abnormalities.

Case # 43 - E.S. - 19/w/m - 1968 Pontiac -GTO - Av-CIR = 1
Rate is 120 per minute
PR: .16
QRS: .08
QT: .32
Sinus rhythm
The mean QRS vector is +100°
T is low to early negative in II, III, aVF, and V7 through VB.
Sinus tachycardia
Primary nonspecific T wave abnormalities.

Case # 44 - M.S. - 50/w/f - 1962 Ford - Av-CIR = 1
Rate is 82 per minute
PR: .14
QRS: .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Case # 45 - J.S. - 39/w/m - 1963 Ford - Thunderbird - Av-CIR = 4
Rate is 100 per minute
PR: .18
QRS: .10
QT: .34
Sinus rhythm

A braod S is present in I, II, aVL and persists in the precordial leads through V6.
RR' is absent over the right precordium.
Occasional ventricular extrasystoles.
Sinus tachycardia

Case # 46 - J.S. - 47/w/m - 1969 Chevrolet - Av-CIR = 3
Rate is 105 per minute
PR: .12
QRS: .08
QT: .32
Sinus rhythm
Sinus tachycardia

Tracing 2 - Rate is 105 per minute
PR: .14
QRS: .08
QT: .32
Sinus rhythm
Sinus tachycardia

Tracing 3 - Rate is 110 per minute
PR: .13
QRS: .08
QT: .30
Sinus rhythm
Complexes are essentially unchanged
Sinus tachycardia

Case # 47 - D.S. - 30/w/f - 1964 Chevrolet - Av-CIR = 5
Rate is 80 per minute
PR: .16
QRS: .08
QT: .36
Ventricular rhythm is regular. Voltage of P is low, and negative in II, III and aVF, positive in aVL.
P waves are not identifiable in the remainder of the leads.
Possible ectopic atrial pacemaker.

Tracing 2 - Rate is 72 per minute
PR: .14
QRS: .08
QT: .36
Complexes are essentially unchanged.
Possible ectopic atrial pacemaker.

Tracing 3 - Rate is 80 per minute
PR: .12
QRS: .08
QT: .36
P waves remain negative in II, III, and aVF.
Ectopic atrial pacemaker.

Case # 48 - S.S. - 18/w/m - 1957 Chevrolet - Av-CIR = 1
Rate is 72 per minute
PR: .14
QRS: .10
QT: .38
Sinus rhythm
Normal electrocardiogram

Case # 49 - C.T. - 32/w/f - 1966 Chevrolet - P/U - Av-CIR = 2
Rate is 94 per minute
PR: .12
QRS: .08
QT: .36
Sinus rhythm
Normal electrocardiogram

Case # 50 - R.T. - 22/w/m - 1966 Ford - Mustang - Av-CIR = 1
Rate is 67 per minute
PR: .14
QRS: .08
QT: .34
Sinus rhythm
Normal electrocardiogram

Case # 51 - L.W. - 22/w/m - 1969 Plymouth - Av-CIR = 1
Rate is 95 per minute
PR: .13
QRS: .08
QT: .32
Sinus rhythm
Normal electrocardiogram

Case # 52 - R.Y. - 11/w/m - 1966 Chevrolet - Corvair - Av-CIR = 4
Rate is 90 per minute
PR: .12
QRS: .12
QT: .40
Sinus rhythm
A broad S is present in I, II, aVL, aVF, and V2 through VB.
RR' is present in V4R through V2
Complete right bundle branch block.
Occasional atrial extrasystoles.

Tracing 2 - Rate is 100 per minute
PR: .13
QRS: .08
QT: .34
Sinus rhythm
QRS interval has decreased and is now normal.
A late S remains present in I, II, III, aVF, and V4R through V6.
Primary nonspecific T wave abnormalities.
Occasional nodal extrasystoles.
The pattern of the right bundle conduction delay remains present but QRS interval is normal.

Tracing 3 - Rate is 122 per minute
PR:
QRS:
QT:
Sinus rhythm
S waves are prominent in I, II and III.
T waves are low to flat in V2 through VB
Sinus tachycardia
Nonspecific T wave abnormalities.

Case # 53 - G.Z. - 62/w/f - 1965 Chevrolet - Corvair - Av-CIR = 3
Rate is 90 per minute
PR: .14
QRS: .08
QT: .34
Sinus rhythm
Voltage of T is low in III, aVF, aVL and V7 through VB.
Minor nonspecific T wave abnormalities.

Case # 54 - T.W. - 32/w/f - 1962 Chevrolet - Av-CIR = 1
Rate is 72 per minute
PR: .14
QRS: .09
QT: .37
Sinus rhythm
Normal electrocardiogram

Tracing 2 - Rate is 60 per minute
PR: .13
QRS: .09
QT: .40
Complexes are essentially unchanged.
Normal electrocardiogram.

APPENDIX E

INJURIES - GROUP I

Case #01 L.B. 61/w/m
Costochondral separation of left ribs.
Comminuted fracture of right radius.
Comminuted fractures - left tibia and fibula.
Facial lacerations.
Comminuted fracture of left femur.
Plateau fracture of right tibia.
Contusions and fractures of right malleolus.
Contusion of pharynx.
Possible fat emboli.

Case #02 J.G. 20/w/f
Cerebral and brain stem contusion.
Fractured pelvis.

Case #03 C.Y. 23/w/m
Depressed fracture of right maxilla.

Case #04 M.W. 21/w/m
Multisystem trauma.
Facial and orbital lacerations.
Contusion of kidney and bladder.
Retroperitoneal hematoma.
Fractures - both legs involving femurs, tibias and fibulas.
Fracture of right foot.

Case #05 W.P. 37/N/m
8 cm. laceration of chin.
Chest pain.

Case #06 R.C. 67/w/m
Fractured right clavicle.
Multiple lacerations.
Fractures of right ribs (3, 4, and 5).

Case #07 H.S. 51/w/m
Contusions of jaw, neck, thorax and abdomen

Case #08 L.F. 38/w/f
Laceration of left knee.
Cerebral contusion.
Multiple lacerations.
Fractured left clavicle.

Case #09 C.W. 21/N/m
Abrasions of the left humerus and both knees.

Case #10 M.C. 64/w/f
Cerebral concussion.
Fractured right clavicle.
Contusions of left mandible.
Laceration of left hand.
Laceration of lower lip.
Cervical sprain.
Abrasions and contusions of both knees.

Case #11 A.M. 47/w/f
General bodily contusions and abrasions.

Case #12 J.J. 35/w/f
General bodily contusions and abrasions.

Case #13 D.G. 20/w/f
Compound fracture of mandible.
Multiple lacerations of extremities and face.
Multiple contusions and abrasions.

Case #14 D.D. 24/w/m
Fractures of cervical vertebrae C3 and C4 with parapalegia.

Case #15 W.H. 40/w/m
Contusions of left chest and abdomen.

Case #16 R.T. 42/w/f
Fractures of left ribs (2-5)
Fracture of left clavicle.

Case #17 B.L. 36/w/f
Fractures of left ribs (3-7)
Fractured pelvis

Case #18 K.J. 16/N/f
No injuries

Case #19 F.M. 24/w/m
Retrograde amnesia

Case #20 E.F. 38/2/m
General abrasions and contusions.

Case #21 N.H. 19/w/f
Contusions of head, chest, and shoulders.

Case #22 G.W. 20/w/m
Massive cerebral contusion.
Facial fractures.

Case #23 P.F. 24/w/m
Cervical sprain
Contusions of pharynx and esophagus.

Case #24 C.B. 20/w/f
Multiple lacerations and contusions.
Fractures - left and right femur.
Fractured left foot (compound)

Case #25 J.J. 42/w/m
Multiple lacerations and contusions.
Contusions of chest with no fractures of ribs or sternum.
Closed fracture - of calcis

Case #26 S.J. 25/w/m
Brain concussion.
Multiple contusions and abrasions.

Case #27 N.B. 50/w/m
Laceration lower leg - 1".
Hematoma of chin and calf.
Laceration of scalp.

Case #28 C.C. 20/w/m
General contusions.

Case #29 C.D. 24/w/f
Compound fracture of left patella
Complete disruption of quadriceps muscles.
Laceration of left ankle.

Case #30 B.A. 29/w/m
No injury.

APPENDIX F

INJURIES - GROUP III

Case #01 G.A. 36/w/m
Fractured nasal bones.

Case #02 W.A. 58/w/m
Fractures of pelvis and ribs.

Case #03 F.A. 34/w/f
Contusions: back, left hip, top of head.

Case #04 G.A. 16/w/m
Contusion of shoulder.
Ruptured spleen.
Fractures of ribs - multiple.
Fracture - mid-shaft of ulna.
Hemothorax secondary to rib fractures.

Case #05 J.B. 43/w/m
Severe facial lacerations.
Fractures - maxilla, zygoma, nasal orbit, frontal sphenoid, ethmoid, and zygomatic arch.
Bilateral laceration of left arm with 5th extensor tendon severed.
Multiple fractures of ribs.

Case #06 R.B. 22/w/m
Mild concussion.
Trauma to rib cage.
Blunt trauma to abdomen.

Case #07 M.B. 16/w/f
Bruised right wrist.

Case #08 L.B. 22/w/m
Cerebral concussion.
Multiple facial lacerations.
Fractured nose.

Case #09 R.B. 20/w/m
Fractured nasal spine of maxilla.

Case #10 H.B. 37/w/m
No apparent injuries.

Case #11 D.C. 19/w/m
1/4" vertical laceration of upper lip.

Case #12 C.C. 20/w/m
Laceration of scalp.

Case #13 L.C. 29/w/f
3 cm. vertical laceration of left forehead.

Case #14 M.C. 36/w/m
No apparent injuries.

Case #15 I.C. 70/w/f
Thru and thru laceration of right nasal ala.
1/2" thru and thru laceration of upper lip.
1" vertical laceration of columella.

Case #16 D.D. 25/w/m
Paraspinus muscle spasm.

Case #17 J.A. 33/w/m
Scalp and facial lacerations.
Cerebral contusion.
Cardiac contusion.
Pulmonary contusion.
Multiple rib fractures.

Case #18 D.E. 16/w/m
Multiple superficial abrasions.

Case #19 J.E. 18/w/f
Facial lacerations.
Fractured mandible.
Loss of 4 teeth.

Case #20 C.E. 15/w/m
3/4" lacerations of both knees.
3-4 cm. laceration of chin.

Case #21 P.E. 18/w/m
No apparent injury.

Case #22 D.F. 22/w/m
Laceration of forehead into frontal scalp.

Case #23 G.F. 75/w/m
Small abrasions on right parietal scalp.
Paraspinus muscle spasm.
Cervical sprain.

Case #24 D.F. 16/w/m
Multiple contusions.
Cerebral concussion.

Case #25 M.G. 16/w/m
Contusion of forehead.
1 cm. laceration of right knee.
Multiple abrasions.
4 cm. laceration of left knee.

Case #26 B.N. 32/w/m
Multiple lacerations of forehead.
Cervical sprain.

Case #27 R.M. 29/w/m
No apparent injury.

Case #28 M.M. 17/w/m
Facial abrasions.
Laceration of right lip.

Case #29 B.M. 19/w/f
Minor contusion of left thigh.

Case #30 C.M. 42/w/m
Cerebral concussion.
Retrograde amnesia.

Case #31 C.M. 18/w/m
Blunt abdominal trauma.

Case #32 K.K. 56/w/m
4 cm. laceration of left temporal area.

Case #33 J.J. 34/w/m
Contusion to anterior thorax and heart.
Multiple lacerations - corner of mouth, chin and right knee.

Case #34 G.J. 17/w/m
Laceration of chin.

Case #35 W.H. 20/w/m
Contusions - left forehead and left knee.

Case #36 S.H. 28/N/m
No apparent injury.

Case #37 T.H. 18/w/m
Cervical muscle spasm.

Case #38 L.H. 27/w/m
Facial and neck lacerations.
Deep laceration of forehead.

Case #39 E.P. 34/w/f
Small laceration outer border of left orbital ridge.
Hematoma left forehead.

Case #40 D.P. 18/w/f
No apparent injury.

Case #41 J.R. 26/w/m
Low back pain and tenderness.

Case #42 B.R. 51/w/m
Contusions of ribs.

Case #43 E.S. 19/w/m
No apparent injury.

Case #44 M.S. 50/w/f
Low back pain.

Case #45 J.S. 39/w/m
Multiple rib fractures.
Multiple contusions.

Case #46 J.S. 47/w/m
Fracture - proximal end of left tibia.
Laceration of tongue.

Case #47 D.S. 30/w/f
Severe facial lacerations.
Closed fracture - olecranon process.
Fracture - distal end of radius.
Pneumothorax.
Multiple facial fractures.

Case #48 S.S. 18/w/m
Slight abrasions - left anterior chest and right knee.

Case #49 C.T. 32/w/f
Scalp laceration.
Cervical contusion.

Case #50 R.T. 22/w/m
Nosebleed.
Contusion of upper lip.

Case #51 L.W. 22/w/m
No apparent injury.

Case #52 R.Y. 11/w/m
Fractures of right and left femurs (right - compound)
Laceration of left leg.
Laceration of tongue.

Case #53 G.Z. 62/w/f
Fracture - left ankle.

Case #54 T.W. 32/w/f
Pain in left chest - No apparent injury.

APPENDIX G

BREAKDOWN OF ELECTROCARDIOGRAMS OBTAINED - GROUP III

Case No.	Subject	ECG #1	ECG #2	ECG #3
1	G.A.	X	-	-
2	W.A.	X	X	X
3	F.A.	X	X	-
4	G.A.	X	X	-
5	J.B.	X	-	-
6	R.B.	X	X	X
7	M.B.	X	-	-
8	L.B.	X	-	-
9	R.B.	X	X	X
10	H.B.	X	-	-
11	D.C.	X	-	-
12	C.C.	X	-	-
13	L.C.	X	-	-
14	M.C.	X	-	-
15	I.C.	X	-	-
16	D.D.	X	-	-
17	J.A.	X	X	X
18	D.E.	X	-	-
19	J.E.	X	X	X
20	C.E.	X	X	-
21	P.E.	X	-	-
22	D.F.	X	-	-
23	G.F.	X	-	-
24	D.F.	X	-	-
25	M.G.	X	-	-
26	B.N.	X	-	-
27	R.M.	X	-	-
28	M.M.	X	-	-
29	B.M.	X	-	-
30	C.M.	X	-	-
31	C.M.	X	-	-
32	K.K.	X	-	-
33	J.J.	X	-	-
34	G.J.	X	-	-
35	W.H.	X	X	-

APPENDIX G--Continued

Case No.	Subject	ECG #1	ECG #2	ECG #3
36	S.H.	X	-	-
37	T.H.	X	-	-
38	L.H.	X	X	-
39	E.P.	X	-	-
40	D.P.	X	X	-
41	J.R.	X	-	-
42	B.R.	X	-	-
43	E.S.	X	-	-
44	M.S.	X	-	-
45	J.S.	X	-	-
46	J.S.	X	X	X
47	D.S.	X	X	X
48	S.S.	X	-	-
49	C.T.	X	-	-
50	R.T.	X	-	-
51	L.W.	X	-	-
52	R.Y.	X	X	X
53	G.Z.	X	-	-
54	T.W.	X	X	-
Subtotals		54	15	8

Totals----------54 Electrocardiograms
15 Serial Electrocardiograms
15 Subjects - At least 2 tracings
8 Subjects - Three tracings